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JEJUNAL ULCER WITHOUT PREVIOUS GASTRO-ENTEROSTOMY

BY EDWARD P. RICHARDSON, M.D., F.A.C.S., BOSTON

IN spite of the extremely active proteolysis which occurs in the upper part of the small intestine during digestion, the region of the bowel from the papilla of Vater to the end of the jejunum is under normal conditions singularly free from lesions arising in the wall of the intestinal tube. Above the papilla, the effect of acid gastric secretion is shown in the common occurrence of gastric or duodenal ulcer; below, as the ileocœcal valve is approached, ulcers from various specific causes, tuberculosis, typhoid fever, dysenteric infections, etc., become increasingly frequent. Surgical lesions of the jejunum, except in so far as they are produced by conditions affecting the intestine from without, for instance, adhesions, herniæ, trauma, the extension of new-growth from adjacent organs or unless they involve the jejunum in common with other parts of the intestinal tract, are almost sufficiently rare to be considered surgical curiosities.

The exception to this general rule at the present time is jejunal ulcer following gastro-enterostomy, which if reported cases are a measure, is probably the commonest lesion of the jejunum today. It has been estimated to occur in about 1.5 per cent of all cases in which gastro-enterostomy is performed. In these cases of jejunal ulcer we have obvious causative factors in the trauma from operation and the action of acid gastric chyme on a part of the intestinal tract normally unexposed to it. In certain cases the persistence of irritating suture material is a further predisposing cause of the jejunal or gastrojejunal ulceration. The ultimate reason, however, why a few cases should develop jejunal ulcer, in spite of all precautions of technique, while the great majority remain free, remains obscure. In recent articles upon jejunal ulcer, an occasional mention is made of the rare occurrence of spontaneous peptic ulcer of the jejunum in individuals in whom no previous operation has been performed. Van Roojen (1), in 1909, stated clearly the possibility of such a spontaneous occurrence of jejunal ulcer. He says that peptic ulcer of the jejunum, analagous to the peptic or round ulcer of the stomach or duodenum, occurs almost always following gastro-enterostomy. Three cases, however, are known to him in which peptic ulcer of the jejunum was found without having been preceded by a gastro-enterostomy. These exceptions to the general rule, not commonly recognized in modern literature, he believes of great importance, since from such cases light may be thrown on the obscure etiology of jejunal ulcer.

The importance of jejunal ulcer following gastro-enterostomy and the obscurity of its more remote causes lends to such primary ulcers of the jejunum an interest not otherwise justified by their extreme rarity. A recent case of spontaneous jejunal ulcer has led me to look through the literature. My hope was that such a review might add something of value in relation to the more common

postoperative type as suggested by Van Roojen. While this hope has not been justified, it has seemed worth while to collect and analyze reported cases of ulcer of the jejunum occurring without previous operation on the stomach. These cases have considerable interest in themselves, since they are obscure both in etiology and in diagnosis, not hard to overlook on the operating-table, dangerous to life, and yet as isolated cases not sufficiently striking to be sure of finding their way into the literature. While on one hand they approach closely in appearance and course to peptic ulcer of the stomach or duodenum, on the other, occasional instances have a resemblance to cases reported as syphilis of the small intestine. My purpose, therefore, is to gather the facts at present available concerning such jejunal ulcers, with especial reference to the recurring statement that peptic ulcer of the jejunum may occur without operation, and to consider the clinical course and etiology of these cases as far as possible from the somewhat meagre evidence at hand.

Ulcers of the intestine, of a type which shows on pathological examination no evidence of a specific causative factor, are not extremely rare in literature. Nor is the idea that such ulcers may be peptic a new one. It is, however, an assumption in regard to etiology for which the evidence is questionable. These ulcers, however, may fairly be classed in accordance with the pathological changes found, which show only a defect, with evidence of necrosis and extension or attempts at limitation and repair, accompanied by inflammatory changes usually slight in degree or by the resulting fibrous tissue. To this extent the pathological picture resembles that of ulcer of the stomach or duodenum, but this resemblance does not necessarily imply a similar etiology. The term usually used for this lesion is simple ulcer, following Cruveilhier, who first clearly described such ulcers occurring in the stomach.

Combes (2), in 1897, collected 36 cases of ulcer of the intestine, which appeared to belong to this simple pathological type. Of these, 7 involved the jejunum, 12 the ileum, 5 the cæcum, 3 the large intestine, and 3 the rectum. Leotta (3), in 1919, collected 22 cases of simple ulcer of the jejuno-ileum, including one personal case involving the ileum. The bowel affected was the jejunum 4 times, the ileum 17 times. While many of the cases reported as simple ulcer are far from conclusive in themselves, yet it remains clear, that after excluding ulceration due to known causes, such as typhoid fever, tuberculosis, new-growth, dysenteric infections, obstructive distention, etc., there remain ulcers of the small intestine, grossly and microscopically of the simple type, to which we cannot assign a definite cause.

The following case I saw in consultation with Dr. L. M. Spear, and sent to the Massachusetts General Hospital believing that his symptoms were due either to perforation of the stomach or to renal stone:

CASE 1. E. S., No. 234,549, male, born in Ireland, age 47, widower. Admitted January 11, 1920, with the following history. In July, 1919, the patient had an attack of severe general abdominal pain, lasting 6 hours, during which he vomited. In September, 1919, a similar attack occurred. There were no gastric symptoms between July and September. After the second attack the patient went to the Boston Dispensary for advice, and was put on a diet which was not followed closely. Since September he has had occasional attacks of transient, but rather severe, pain localized in the left abdomen. For the past 2 weeks he has noticed that his bowel movements have been black although no medicine has been taken except epsom salts and cascara.

Yesterday the patient was seized with such severe abdominal pain in the left lower quadrant, radiating to the left flank and shoulder, that he rolled on the floor. He was given morphia and was able to sleep a little. The pain has persisted. He vomited once last night, and repeatedly this morning. His bowels moved many times yesterday but not today.

Patient has never had any gastric trouble up to July, 1919, but has always been more or less constipated, for which he took salts. There have been no cardiac or respiratory symptoms. He has had gonorrhœa several times, the last about 8 years ago when a bubo was incised in the left groin. His genitourinary history was otherwise negative.

Patient is a well developed and nourished man, looking rather weak, evidently suffering some pain but not in shock. Examination of skin is negative. There is a scar 3 centimeters long in left groin. The pupils react to light and distance, are equal, and movement is normal. The ears and nose are negative. The mouth shows pyorrhœa and sordes. The throat, tonsils, and neck are negative. The thorax is well developed, expansion is equal, the lungs clear and resonant. The heart shows no enlargement, displacement, murmurs or thrills.

The abdomen was tympanitic throughout, the tympany extending 3 centimeters above the costal margin; the liver dullness reaching from this point to the sixth intercostal space. The patient held his abdomen tense; no masses or herniæ were palpable. The left side was rigid, and there was tenderness about 9 centimeters below the left costal margin and in the left flank. There was spasm over this area of tenderness.

Rectal examination was negative. Reflexes were negative.

Temperature 98.8°; pulse 82; respiration 20.

White count, 18,400; Blood pressure 140–90.

Urine examination negative.

X-ray for renal or ureteral stone was negative.

Operation 3:20 p.m., January 11, 1920. Ether. Cloudy fluid was found on opening the peritoneal cavity through the upper left rectus muscle. A perforation of the stomach or duodenum was looked for, but these organs were negative. A small mass was then felt in the bowel, slightly adherent to the posterior abdominal wall, in contact with the splenic flexure. On delivery this proved to be small intestine. The induration was due to a stricture, on the anterior surface of which was a perforation ⅛ inch in diameter. At first attempt was made to close this perforation by excision and suture similar to Heinecke-Mickulicz pyloroplasty. On opening the gut, however, an annular ulcer encircled the bowel at the point of stricture, through which perforation had taken place, and the amount of constriction and induration made a plastic procedure impossible. An atypical resection was done, removing the ulcer and its margins, without the adjacent mesentery, followed by an end-to-end anastomosis. A resection of the whole loop would have proved easier. The level of the stricture was not exactly determined on account of the patient's condition. It was believed to be within 2 feet of the end of the duodenum. Closure was without drainage.

Pathological report by H. F. Hartwell. A review of the microscopic sections from the jejunum shows no evidence of syphilis. The pathological examination is therefore as follows:

A number of irregular fragments, the largest of which contains part of an ulcer in its mucosa. Its edge is slightly raised and its base is rough. At one portion of the base there is a small perforation passing directly through the wall of the gut. Its peritoneal surface is covered with plaques of fibrin.

Microscopic examination shows an ulcer, whose surface is covered with necrotic exudate. Its base is formed by fibrous tissue which extends through the entire thickness of the wall. There is considerable inflammatory exudate on the peritoneal surface. There is no evidence of tuberculosis or malignant disease.

Sections stained by Levaditi's method show no spirochæta pallida.

Diagnosis: chronic ulcer.

The patient made a good recovery without complications, and was discharged on February 3, 1920.

His Wassermann reaction is not recorded.

X-ray February 5, 1920, after operation: Stomach is in medium height but pyloric end appeared further displaced to right than normal. No visible abnormality of filling or outline is seen. It was empty in normal time. The sphincter appeared regular and the first part of the duodenum filled regularly. It appeared oblique in position as though drawn to right. The ileum was not remarkable. Barium had not entered the cæcum at 6 hours and was in the rectum in 24 hours when cæcum was empty and colon was outlined by traces of barium only. The only abnormality noted was the position of the stomach and duodenum which is suggestive of adhesions in the right abdomen. No definite lesion was found.

On searching through the records of the Massachusetts General Hospital, only one similar case was found. For the privilege of reporting it, I am indebted to Dr. C. A. Porter:

Case 2. W. S., 181,349, female, age 48, married, born in Russia. Admitted March 3, 1912, with diagnosis: intestinal obstruction, peritonitis. The family history was negative for tuberculosis, cancer, and hæmophilia.

Patient has never been in a hospital except in confinement. She has always had fair health except for constipation and stomach trouble. For years she has been subject to pain in the heart after eating, and has vomited a great deal, but does not know the color of the vomitus. She has had no cardio-respiratory symptoms. The so-called pain of the heart (epigastrium) has radiated to the shoulders. She has had two children and one miscarriage. Catamenia not remarkable.

Patient has been indisposed for about 7 weeks. A week ago she began to have severe pain about the heart and vomited "awful." Since that time she has been in bed and has vomited occasionally every day. Her bowels have moved with small result. She has seen nothing remarkable in the stool, and has noted no blood in the vomitus. Repeated enemas have failed to give anything like good results. A note from her doctor states that she began to have severe abdominal pain this morning and repeated attempts failed to move the bowels.

Physical examination showed a well-developed and stout Jewish woman, lying in evident distress. The pupils react. The tongue is coated; the mouth otherwise negative. Examination of chest shows hyperresonant note. The breath sounds are clear except at base where there were numerous deep râles. The heart is not enlarged; sounds regular, fair quality, clear. The abdomen is full, rather distended, but not rigid. There were numerous blistered areas over epigastrium, the result of turpentine stupes. No masses are palpable, but tenderness is present through the whole abdomen, more particularly in the epigastrium. No visible peristalsis is observed.

Rectal examination shows nothing except increased abdominal tenderness.

Knee jerks are present; no œdema.

Temperature 98°; pulse, 112; white count, 19,000.

Operation, March 4, 1921, by Dr. C. A. Porter. Ether. On median suprapubic incision, thin, cloudy fluid and fibrin flakes without odor were found. Intestines were distended and injected. Exploration shows a mass in the right upper quadrant. A transverse 5-inch incision was then made along the right costal margin. The tumor was brought into the wound and was found to be a loop of jejunum 6 inches long, twisted, and kinked, and bound down by adhesions. On freeing the adhesions, fæcal matter escaped from a perforation in the gut. No attempt was made to locate the portion of gut which was involved, but it seemed to be the middle of the jejunum. The bowel on one side of the kink was collapsed, on the other distended. A loop, 6 inches in length, was resected and an end-to-end anastomosis done with Pagenstecker. The transverse incision was closed with drainage of the superficial fascia. A rubber tube was left in the pelvic wound.

Pathological report. The specimen is a piece of intestine 15 centimeters long. The serous surface is injected and covered with fibrin. Section shows the walls and mucosa considerably darkened with a soft, moist appearance. A ragged, circular perforation through all coats 1½ centimeters in diameter is present in one extremity of the gut. The intestine on one side of the ulceration 8½ centimeters, on the other side 2½ centimeters in transverse diameter.

Microscopic examination of the edge of the ulcer showed a little granulation tissue but no evidence of any new-growth. The blood vessels in the immediate vicinity were not especially altered or thrombosed.

Diagnosis: Simple ulcer.

Discharged, March 23, 1912.

In neither case was the level of the lesion exactly determined. It seems fair, however, if the ordinary arrangement of the intestinal coils held good in these instances, to accept the operative diagnosis of ulcer of the jejunum level undetermined, although in Case 1 the loop involved was probably not far below the end of the duodenum. In neither case is the Wassermann reaction known, although syphilitic exposure would seem probable in Case 1, on account of the repeated attacks of gonorrhœa. Sections of the ulcer, however, were negative for spirochæta pallida by the Levaditi method. There was nothing suggestive of tuberculosis on microscopic examination in either case. Each presents a chronic ulcer of the jejunum which cannot be assigned to the usual causes of intestinal ulceration. In their pathological findings, chronicity, induration, tendency to cicatricial contraction, and ultimate perforation, they have much in common with peptic ulcer of the stomach or duodenum.

The records of 4,200 autopsies in the Pathological Department of the Massachusetts General Hospital, examined by permission of the director, Dr. J. H. Wright, shows no case directly comparable to the above. Further evidence, therefore, depends upon the literature. I have been able to find the following cases, nearly all incomplete in various respects, abstracts of which I present at this point, in order to facilitate a discussion of the general aspects of jejunal ulcer arising without operative interference. Cases from the older literature have been included, since the total number is so few.

Case 3. Reported by Wagner (4), in 1858. A laborer of 18 was buried up to the lower chest by a fall of earth. He gave evidence of peritonitis and died 22 hours later. Autopsy showed diffuse peritonitis. The mucosa of the stomach and duodenum was normal. After freeing omental adhesions, on the surface of the jejunum, opposite the mesentery, 6 inches from the end of the duodenum, an oval hole was found, from which intestinal contents flowed. The adjacent mucosa showed normal microscopic structures. The remaining intestine showed no ulceration. A reconstruction of the anatomical changes is most probably as follows: a chronic ulcer of the upper jejunum gradually consuming all layers was hindered, at least temporarily, from perforation by adhesions of the omentum. As a result of burying, the omental adhesion was separated. The jejunal ulcer had the greatest similarity with the so-called perforating or simple chronic ulcer of the stomach, and is to the same, at all events, analogous.

Case 4. Reported by Taylor (5), in 1865. A man, age 45, given to drinking, who after drinking beer freely and being thrown in a wrestling match complained of having strained himself in the abdomen. He walked home, a distance of a mile, and complained of intense pain from time to time until his death, which took place 50 hours after the accident. The postmortem showed evidence of a severe peritonitis, and a small aperture, which would not admit a pin-head, was found in the jejunum. This aperture seemed to have been the result of an ulcer, which had commenced on the outer surface of the intestine.

Case 5. Reported by Perroud in 1866 (abstract from Combes (2). A cooper, 32 years old, in good general health, suffered for 3 months from slight dyspepsia. While at stool he felt a violent colic, soon followed by three periods of syncope. The pain continued; symptoms of peritonitis and obstruction developed, death occurring about 36 hours after onset. Autopsy showed acute diffuse peritonitis. No

intestinal obstruction was found, but in the right hypochondrium in a loop of small intestine, near the end of the duodenum, a small rounded opening was demonstrated the size of a lentil, situated at the level of the mesenteric insertion, communicating with the interior of the intestine, and explaining the presence of gas and fæces in the peritoneal cavity, as well as the acute peritonitis. The walls of the intestine, in the neighborhood of the perforation were red, a little thickened, and much softened. The remainder of the digestive tube was entirely sound. No alteration was found in the other organs. Tubercles were nowhere in evidence.

This case appears to be an example of simple ulceration of the small intestine, which one can compare with those ulcerations of the stomach which develop without much inflammation, and which only show themselves during life by very slight symptoms of dyspepsia.

The author does not state definitely whether the perforation was above or below the end of the duodenum. From the terms of the description, and the presence of fæcal matter in the abdominal cavity, it seems probable that the jejunum was involved.

CASE 6. Reported by Reverdin (6), in 1867. A man of 55, of alcoholic habits, died after 2 days of vomiting and obstipation. Autopsy showed evidence of peritonitis. On the right, halfway between the iliac crest and the liver, was an intestinal perforation. The stomach and duodenum, very slightly dilated, presented no alteration of their mucosa. The mucosa of the intestine was normal throughout, except at the level of the perforation. The latter was situated on the free border of the intestine, at the level of the middle part of the jejunum. It was almost round, the size of a 50-centime piece. Its margins, shaped as by a punch, were a little thick, and turned back from the serous side. No foreign body was present in the intestine. The remaining organs were essentially negative. No lesion of the abdominal aorta was present.

CASE 7. Reported by Simpson (7), in 1897. A man of 56, by trade a maltster, whose mental condition at death was one of dementia consecutive to chronic mania, collapsed and died suddenly. He was accustomed to devour filth and rubbish of all sorts. He had complained only of slight pain and constipation. Autopsy showed no marks of injury. The peritoneal cavity contained about 30 ounces of blood-stained serum, but no fæcal matter. The mucous membrane of the alimentary canal was normal, except at a point 6 inches below the beginning of the jejunum, where there was a single ulcer about 6 millimeters in diameter, irregularly circular in outline and with clean-cut edges which showed no signs of chronicity, perforating through all its coats. A few shreds of wood fiber were the only foreign bodies in the alimentary tract, and no extraneous substance was discovered in the peritoneal cavity. The gall bladder contained stones. The heart showed hypertrophy and dilatation; there was well-marked degeneration of the myocardium: the aortic valves were somewhat thickened. Both kidneys were in an advanced condition of interstitial nephritis.

The lesion of the jejunum was regarded by those present as being the homologue of perforating gastric ulcer and the acute ulceration of the duodenum which is often found as a sequence of severe burns or scalds, but there seems a possibility that it may have originated by injury of the intestinal wall by some foreign body introduced by the mouth.

CASE 8. Reported in Brit. M. J., 1906, i, 718—a medico-legal case. A man of 57 who had been under treatment for heart disease, hydrocele and stricture, on the morning of February 10, while carrying a tray and passing through a door, ran against the corner, hurting himself. He died suddenly at 1 a.m., February 12. At the postmortem examination, a bruise was seen in the left groin and a slight bruise on the internal abdominal wall corresponding with the bruise on the surface. The bruise was about a foot from the spot where the ulcer of the intestine had ruptured. Death proved traceable to a perforation of a small chronic ulcer of the small intestine, 8 feet from the stomach, through which a few drops of fæcal matter had percolated, and there were signs of commencing peritonitis.

CASE 9. Schmilinski (8), in 1910, reported a case of stenosing ulcer of the jejunum. The patient, a woman of 63, had for years pain in the stomach, with on two occasions vomiting of blood and malæna. Findings, 3 years ago, obstructive incompetence of stomach with residue; hypersecretion. Diagnosis: stenosing ulcer of the pylorus or duodenum. Until January, 1909, she was free from pain by means of diet and lavage, when a recurrence of the pain developed. In the fasting contents, no free hydrochloric acid was found, instead of which was an admixture of bile. Air blown into the stomach emptied itself into the duodenum remarkably quickly for a pyloric stenosis.

At operation the duodenum and beginning of the jejunum were found dilated. Four centimeters below the duodenojejunal fold was a stenosing induration of the jejunum and adjacent mesentery. Resection; recovery. The specimen showed on the mesenteric border of the stenosis a small ulcer, which, as well as its surroundings, showed microscopically the signs of chronic inflammation.

In the discussion, Fraenkel had observed a similar case, in which he arrived at a diagnosis of syphilis by means of staining the elastic tissues. Again, in a case of phthisis, he found a stricturing ulcer at this point. The present case appeared to him syphilitic. Katz raised the question whether at the examination of the patient 3 years previously, a pyloric in-

competence might not have already existed. In this case it was possible that as a result of the existing hypersecretion a peptic ulcer might have developed, which had led to a stenosis.

Schmilinski, in closing, said that at the previous examination, whether or not the pylorus was capable of closing was not determined. The previous statements gave great weight to the acceptance of a syphilitic etiology for the ulcer. The elastic fibers were not investigated. The remaining findings gave nothing of note either for syphilis or for tuberculosis.

CASE 10. Cade (9), in 1913, reported the case of a man, age 25, a grocer clerk; who entered hospital June 23, 1912; died June 24, 1912. His health was habitually good. He denies excessive drinking and syphilis. A month ago, he had moderately severe pain in the left iliac fossa, relieved by medicine, so that he was able to continue his work. Ten days ago the pain returned. A large mass was found at this time in the left iliac fossa. He then improved somewhat and was able to come on foot to the hospital. At 8 p.m., shortly after his arrival, he felt a violent, stabbing pain in the lower abdomen. Copious vomiting, complete obstipation, hiccough and the development of great distention ensued. Collapse and death occurred the next morning. Autopsy showed the abdomen full of pus. In the left iliac fossa the intestine presented a perforation; pressure forced out the contents. The perforation was situated in a loop of small intestine adherent to the pelvic colon. On dissecting the intestine, nothing abnormal was found in the large intestine or appendix. In the small intestine, about 60 centimeters below the duodenum, the affected loop was found. At this point there was a thick, circular zone formed of fibrous tissue, about 1 centimeter in breadth, which, however, did not cause a stenosis. Immediately above this zone there was a blackish, necrotic area, and it was at the middle of this that the perforation was situated, about 5 millimeters in diameter, with ragged edges. The remaining small intestines were negative, as were the kidneys, heart, and lungs. Many mesenteric glands were hypertrophied. Microscopic examination of the edge of the ulceration showed thickening of the intestinal wall, inflammatory infiltration, and necrosis of the mucosa. No change was found suggestive of tuberculosis. A section of the mesentery showed the vessels permeable; of a mesenteric gland, recent inflammatory changes.

CASE 11. Murphy (10), in 1916, reports the case of ulcer of the duodenum and jejunum. The patient, a man of 62, whose family and past histories were negative, had suffered for 15 years with digestive disturbances, worse during the past 4 years, with a tendency to periodic attacks, about once a month. The principal symptoms have been pain in the epigastrium, and vomiting. Six weeks ago, there was evidence of near perforation in the upper abdomen followed by abdominal soreness and tarry stools. A few days later hæmatemesis occurred. Roentgenograms revealed obstruction to the outlet of the stomach and involvement of the gall bladder. The diagnosis was duodenal ulcer with attempts at perforation and sequential adhesions.

Operation: "As we explore the upper abdomen, we find the gall bladder empties itself easily, but is adherent to the duodenum. The upper surface of the arch of the duodenum is involved in an extensive inflammatory induration which indicates the site of a duodenal ulcer. . . . There is another ulcer in the first part of the jejunum. This is but the second ulcer I have seen in this situation."

Anterior gastro-enterostomy was performed. The patient was reported as much improved 3 months later.

CASE 12. Bryan (11), in 1916, reports the case of a man, age 48, who 25 years previously had typhoid fever, and 2 years later a mastoiditis, which resulted in unilateral deafness and a sinus still persisting. He never drank or used tobacco. In the last 4 years he has had severe attacks of indigestion with increasing frequency of late, characterized by violent cramps in the region of the stomach. Two years ago he had constant pain and nausea, which continued for 4 months. He vomited constantly, but never noticed any blood. A diagnosis of ulcer of the stomach was made. The pain and nausea, although somewhat relieved by gastric lavage, have persisted, so that he has been unable to do any regular work for three years. September 8, 1915, at 11 a.m., he was seized with agonizing pain in the abdomen and groin. At 8 a.m. the picture was that of acute abdomen and the diagnosis of probable perforation of ulcer of the stomach was made. Operation was refused until 8 p.m. The peritoneal cavity was full of greenish fluid with flakes. The stomach was atrophic, hard, bound down, pulled to the left, and firm, the walls white and heavy, the omentum shrunken and thick. The duodenum was plastered down, the jejunal wall was likewise indurated, whitish, and thickened; and on its anterior wall, about 3 inches from the duodenojejunal junction, a round, punched-out ulcer, the size of a cherry stone was found. The induration of the jejunum extended several inches below the point of perforation and tapered off gradually in the jejunal wall. Closure of the perforation with omentum. Death. No autopsy.

These cases, with the two reported in this paper, make the whole basis, so far as I have been able to find it, for a discussion of the spontaneous occurrence of peptic ulcer in the jejunum. Some of the cases are so incomplete as to be practically worthless, yet as a whole they form a clinical group with a good deal in common, and as far as the evidence goes, seem to be instances of simple ulcer. My aim has

been to be inclusive rather than critical. Only one case is confirmed both by microscopic examination and autopsy, only three others by microscopic examination and of the six remaining autopsies, several are very briefly reported.

Of the 12 cases, 9 occurred between the ages of 45 and 63, and 10 were in men. In five autopsies in which the condition of the whole bowel is mentioned, the jejunal ulcer was the only one present in the intestinal tract, excluding for these cases a general ulcerative process. In the remaining cases, only one intestinal ulcer was actually demonstrated although more might exist. In 6 cases, confirmed five times by autopsy, and once, in my own case, by operative evidence and X-ray, the stomach and duodenum were apparently negative. Two cases, those of Murphy and Bryan, showed a lesion of the duodenum or stomach. In four cases the condition of these organs is uncertain. The most frequent situation for the ulcer was within a few inches of the end of the duodenum, occurring there five times. The other ulcers lay between this point and the middle of the jejunum, or possibly lower. The ulcer in 7 cases showed evidence of chronicity, and was associated three times with a definite stricture of the bowel. In 2 cases the ulcer seemed acute, in the remaining case the point is not clear. Perforation occurred ten times in the 12 cases.

Description of the ulcer itself is rather vague. Three times its general resemblance to ulcer of the stomach is noted, twice more its punched-out character is mentioned. Only once was it apparently annular; in general it appeared to affect a limited part of the bowel circumference. In the 3 cases with constriction of the bowel it is hard to say whether the constriction was the effect or the cause of the ulcer, although the former seems to be the case. In 1 case (Cade) the perforation took place above a zone of fibrous tissue in the intestine.

The microscopic examination in 4 cases showed the changes associated with simple ulcer, without evidence pointing toward a definite cause, infectious or otherwise. In only 1 case did this examination include the mesenteric glands which showed only inflammatory changes. These findings are of some negative value in excluding tuberculosis, but are of little positive help in connection with the cause of the ulcers. One case examined by the Levaditi method showed no spirochæta pallida. In no case is the Wassermann reaction known.

Since some of these ulcers are definitely chronic in course, it is conceivable that they might prove a starting point for the rare cases of cancer of the jejunum. I am unable to find evidence for this point. Herzog (12) reports a case in which two primary cancers, one of the ileum and one of the ileocæcal valve, developed on the site of old tubercular ulcers.

Little ground exists for a discussion of the etiology of these ulcers either as a group or as individual cases. In two cases alcoholism, in one chronic dementia, filthy habits of eating, and interstitial nephritis, in another stricture of the urethra and heart disease, may be contributing factors; they hardly suffice as a complete explanation for the occurrence of the jejunal ulcer. In one case (Schmilinski) syphilis might have been an adequate cause. But as a whole we can only say that there are certain types of intestinal ulceration to which the majority at least of these cases do not belong. Ulcers of a dysenteric nature may be excluded, since such cases would show a diffuse ulcerative process lower down in the intestinal tract, a condition excluded by the cases coming to autopsy, and not suggested by the clinical history or operative findings in the remaining cases. Nor is it likely that these cases, with as far as known a single destructive lesion high in the intestinal tract, belong to the rather indefinite groups of catarrhal, follicular, and toxæmic intestinal ulceration. Further the evidence does not favor a recent gross embolism or thrombosis.

Active typhoid fever may be excluded as a cause. While typhoid ulcers may involve the jejunum, and even perforate it, they are rare at this level. Further, typhoid ulcers usually heal without cicatricial contraction, and would be unlikely to leave an area of lowered resistance in the jejunum.

Instances of perforation of the small bowel due to stasis and distention above an obstruction are not rare. Such cases have not been

included when recognized since the cause of the ulceration seemed definite. In Case 2 this cause should be considered but here the ulcer occurred at the point of greatest constriction, and not above it. In Cade's case, where the perforation took place above a zone of fibrous tissue, there was no stenosis.

Strangulation of the bowel may lead to cicatricial stenosis. Intestinal ulceration and perforation may occur above or at such a stricture. Sestier (13) in 1827 reported a case of perforation of the jejunum occurring from this cause following a successful operation for strangulated hernia. Meyer (14) has shown that a stenosis persisting after strangulation of the bowel is not necessarily double, although the intestine is commonly constricted at two points. In a review of the literature he found that a stenosis might develop either from the whole incarcerated loop, forming a relatively long narrowing, or only from the parts of the loop of bowel actually constricted by the hernial ring. In the latter case, single and double narrow stenosis were reported in the literature with equal frequency. There is no reason for suspecting a stricture due to previous strangulation as the cause of jejunal ulcer in the present series of cases; it must, however, be considered a possibility.

Active tuberculosis as a cause of the ulcers seems improbable, since in most cases it would be recognizable on gross examination. Tuberculosis of the jejunum alone is rare. In 49 cases of tubercular ulcers of the small intestine, found among 4,200 autopsies at the Massachusetts General Hospital, the ileum was involved 43 times, the jejunum and ileum 5 times. One child of 2, dying of generalized tuberculosis, showed a few groups of non-ulcerated tubercles in the jejunum only. Gross perforation of the ileum into the peritoneal cavity occurred 3 times. The fact that perforation of a tubercular ulcer is unusual, occurring in only from 1 to 5 per cent of all cases, according to Cruice (15), is hardly an argument against tuberculosis in such a rare lesion as the one under discussion.

In the case of a healed or nearly healed tubercular lesion of the intestine, resulting in a stricture or an area of lowered resistance, the primary cause might not be found on gross examination, and might even be overlooked microscopically, if the mesenteric lymph glands were not examined. Yet such an area might be subject to secondary ulceration from sepsis or irritation. That this is possible is shown by a case of Lazerevic (16), where a stenosing chronic ulcer of the jejunum led to diffuse peritonitis, the tubercular nature of the lesion becoming apparent on microscopic examination. It is undeniable that some of these jejunal ulcers may have been tubercular in the beginning, but this does not satisfactorily explain the group as a whole, particularly since the cases coming to autopsy do not record tuberculosis in other organs.

The surgical importance of syphilis of the stomach is well recognized. It is less generally appreciated that syphilis of the small intestine may cause surgical lesions. Both congenital and acquired syphilis may affect the small intestine. The former is less rare, being observed at autopsy in infants dying of hereditary syphilis, and is not of immediate importance. Obendorfer (17) in 1900 collected 24 cases of acquired syphilis of the intestine, in 5 cases associated with syphilis of the stomach. The small intestine was affected in 18 cases, in half of which the jejunum or jejunum and ileum were involved. Perforation occurred twice. Weiss (18), in 1902, found that perforation occurred seven times in 35 cases of syphilis of the intestine. Fraenckel (19), in 1910, reported 3 cases of acquired syphilis of the small intestine, situated twice in the jejunum and once in the ileum, the only ones observed in a material of about 19,000 autopsies. He adds 4 other cases of possible syphilis of the small intestine occurring in the literature since 1900. He believes that there has been much confusion in the past between tuberculosis and syphilis of the intestine. Even admitting that many of the cases previously described as syphilis may have been partly healed tuberculosis, or intestinal ulcers or strictures due to other causes, the occurrence of syphilitic ulcers and strictures of the small intestine seems certain. The presence of spirochæta pallida has been shown in acquired syphilis of the small intestine by Arkin (20) and in congenital syphilis by various observers.

MacCallum (21) describes the lesion of acquired syphilis as follows: "Tertiary lesions of the small intestine are usually localized in the jejunum, where they appear as flat elevations of the character of syphilitic granulation tissue involving mucosa and submucosa. Multiple ulcers are formed, which extend in the forms of rings around the gut and which in healing may produce strictures."

The proof that a lesion of the small intestine is syphilitic is not easy. It may be suggested clinically by the history, by the effect of antisyphilitic treatment, or by the Wassermann reaction. The gross appearance of the intestinal lesion may be suggestive and may be supported by syphilitic lesions in other organs. Similarly the microscopic examination may be strongly suggestive of syphilis, particularly in the presence of endophlebitis and endarteritis with gummatous infiltration. But there is no conclusive point by which the nature of the syphilitic lesion may be recognized in all its stages unless the spirochæta pallida is demonstrated in the lesion. Conversely, the exclusion of syphilis as a cause of intestinal ulceration is correspondingly difficult.

For a consideration of syphilis, the evidence in the present series of cases is lacking. It is true that in no case except Schmilinski's there is a history suggesting syphilis, but in no case is the Wassermann reaction known. The gross lesion described as syphilis of the small intestine, girdle-like constricting ulcers, usually multiple, occasionally with diffuse infiltration of the bowel wall, do not correspond with the lesion described in these cases, with the possible exception of Case 1, Schmilinski's and Bryan's. We can only say that syphilis produces a type of ulceration, leading to stenosis and occasionally to perforation which in contradistinction to other types of intestinal ulceration, affects the jejunum more frequently than other parts of the small intestine, and thus forms an exception to the general rule of the higher resistance of the jejunum to pathological changes. Whether some of the cases included as simple ulcer of the jejunum are syphilitic in origin can not be stated.

It is only after the exclusion of the causes previously mentioned that the question whether these ulcers of the jejunum should be classed as peptic ulcers can fairly be discussed. I take peptic ulcer to mean, in the usual clinical sense, an ulcer in the production of which the acid gastric secretion is an important factor, the remaining factors necessary for ulcer formation being not yet entirely clear. In the 5 cases in which the ulcer occurred close to the end of the duodenum, it is possible that the acid gastric juice may have been a factor. In the 2 cases with an associated lesion of the stomach or duodenum, the ulcer was in this situation. In these cases it is possible that interference with pyloric control, through cicatricial involvement or otherwise, might permit a discharge of gastric contents in such a fashion that neutralization in the duodenum did not occur. In the cases without a lesion near the pylorus, persistence of the gastric acidity in the jejunum is harder to imagine. As for the remaining cases, occurring lower in the jejunum, their position alone would seem to exclude acidity as a possible factor.

To explain ulcers lower in the jejunum, the question of a digestive ulcer from alkaline jejunal contents arises. While such a condition is conceivable, particularly secondary to a pre-existing lesion, the mass of clinical experience is against this assumption and it can only be considered a possibility for which confirmatory evidence is lacking. It seems clear, however, that these jejunal ulcers as a whole cannot be classed as peptic ulcers in the usual clinical sense.

The etiology of these cases, therefore, remains in doubt. Nor is it at all clear that they should be considered a group with a common cause. Their clinical resemblance may well be due more to their situation than to their etiology. The usual clinical course is a sudden peritonitis from perforation without previous warning, as occurred six times, associated in three instances with more or less trauma. In one case perforation followed a definite attack of localizing peritonitis. Four cases gave upper abdominal symptoms more or less suggesting gastric or duodenal ulcer. In two of these a definite gastric or duodenal lesion existed. One case only gave symptoms which might suggest the lesion. Here the symptoms suggested pyloric obstruction, but air blown into the stomach emptied itself into the duo-

denum remarkably quickly, and bile was found in the fasting contents, thus suggesting an obstruction below the duodenal papilla. The only possibility of diagnosis previous to perforation would appear to depend on the demonstration of a jejunal stenosis by clinical or X-ray evidence.

Five cases were operated on; in 3, the jejunum was resected, in 1 a gastro-enterostomy was done for associated duodenal ulcer; these cases recovered. One case of advanced peritonitis with closure of the perforation with omentum died. The remaining cases died without operative interference. The operation of choice, on account of the danger of perforation, appears to be resection of the bowel or possibly excision of the ulcer. Short-circuiting, by gastro-enterostomy, or otherwise, may be necessary for a stenosing ulcer, in which resection is not possible, or when there is associated disease of the stomach or duodenum. In desperate cases, closure of the perforation, by the means most readily available, is the obvious course.

To sum up, the literature gives evidence of the rare occurrence of jejunal ulcer apparently of simple type, resembling somewhat peptic ulcer of the stomach or duodenum, and having a similar tendency to perforation and occasionally to cicatricial stenosis. No sharp line can be drawn between these simple ulcers of the jejunum, and similar ulcers occurring lower in the small intestine. While for the purposes of this paper jejunal ulcers only have been considered, it would seem more rational to discuss as one inclusive group, simple ulcers of the jejuno-ileum. Whether these represent a clinical entity, or are merely the odds and ends of various pathological processes, can not be asserted.

The two cases of jejunal ulcer reported in this paper appear to be instances of simple ulcers affecting the jejunum. Such ulcers occur chiefly in males beyond middle life. No definite etiology may be assigned to them. It is possible that in so rare a condition there may be almost as many causes as cases. In the cases where jejunal ulceration occurs close to the end of the duodenum, it is possible that disturbance of pyloric control may lead to gastric contents reaching the jejunum without neutralization, and so to the formation of true peptic ulcer. For ulcers situated lower in the intestinal tract, this cause seems improbable. Since syphilis produces a form of intestinal ulceration which affects the jejunum more readily than the ileum, and also has a tendency to cicatricial contraction and perforation, it must be considered as a possible cause of ulceration in some of these cases, although definite confirmatory evidence is lacking. Two points are of particular importance in future cases: a careful microscopic study of the lesions, including if possible the mesenteric lymph nodes, and the determination of the presence or absence of syphilis. Whatever the cause, the occurrence of perforation gives these ulcers a definite surgical importance, the more so since the perforation does not take place in one of the regions in which we are accustomed to search for it. The present summary is merely offered to call attention to this type of ulcer and the possible relation of some of these cases to syphilis of the small intestine.

BIBLIOGRAPHY

1. Van Roojen. Arch. f. klin. Chir., 1909, xci, 381.
2. Combes. Les ulcères, simple de l'intestin. Thèse de doct., Toulouse, 1897.
3. Leotta. Arch. ital. di chir., 1919–1920, i, 349.
4. Wagner. Arch. f. Physiol. Heilkunde, 1858, ii, 280.
5. Taylor. Guy's Hosp. Reports, 1865, xi, 282.
6. Reverdin. Bull. et mém. Soc. anat. de Par., 1867, xii, 597.
7. Simpson. Practitioner, Lond., 1897, lviii, 165.
8. Schmilinski. Muenchen. med. Wchnschr., 1910, lvii, 1616.
9. Cade et al. Progrès méd., 1913, No. 24, 309.
10. Murphy. Surg. Clin., 1916, v, 431.
11. Bryan. Surg., Gynec. & Obst., 1916, xxii, 279.
12. Herzog. Beitr. z. path. Anat. u. z. allg. Path., 1912–1913, lv, 177.
13. Sestier. Bull. et mém. Soc. Anat. de Par., 1827, iii, 33.
14. Meyer. Deutsch. Ztschr. f. Chir., 1905, lxxvi, 297.
15. Cruice. Am. J. M. Sc., Phil., 1911, cxlii, 683.
16. Lazerevic. Deutsch. med. Wchnschr., 1919, xlv, 123.
17. Obendorfer. Arch. f. path. Anat., etc., Berl., 1900, clix, 179.
18. Weiss. Zentralbl. f. d. Grenzgeb. d. Med. u. Chir., 1902, v, 577.
19. Fraenckel. Arch. f. path. Anat., etc., Berl., 1910, 131.
20. Arkin. Tr. Chicago Path. Soc., 1909–1912, viii, 224.
21. MacCallum. Textbook of Pathology, 1916, p. 656.

THE SURGICAL TREATMENT OF PERFORATED ULCERS OF THE STOMACH AND DUODENUM

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SINCE the time of Mikulicz perforation of a gastric or duodenal ulcer has been an indication for laparotomy. The several methods practised have had but one purpose—closure of the perforation. One was content with this simple procedure, which consisted in suturing, covering the defect with omentum, and fixing the ulcer to the anterior abdominal wall. The operator did not do more than excise the ulcer, for he did not trust himself to carry out a more radical measure. Kausch, even as late as 1913, distinctly warned against a more radical operation. To my recollection, Haberer, in 1919, was the first to report successful resections in cases of perforated ulcer of the stomach. Haberer was followed by Eunicke and Massari, who reported similar cases. The first complete resections were carried out, in 1920, by Schwarzmann of our division. He reported 9 cases of resection of the stomach, 8 of which resulted in cures. Since then only 6 cases have appeared in the literature. One was reported by Henry, of the Montreal General Hospital, in Surgery, Gynecology and Obstetrics, 1921, xxxii. Henry described a case of recurring perforation. His article has prompted me to report our conclusions which are based on an enlarged experience in treating, by means of resection, perforated gastric and duodenal ulcer.

One advantage of the conservative methods is that the operation consumes only a short time. On the other hand, prognosis can only be indefinite for such methods do not remove the ulcer. By means of gastro-enterostomy, however, one endeavors at least to heal. Does this really help the patient?

At the surgical congress in 1905, reports from many sources were made, to the effect that gastro-enterostomy did not always yield functional results. After operation one observed at first a disappearance of the complaints due to the ulcer—a lowering of the hyperacidity, even a return to normal acidity —but this was not true in all cases, for there began to appear recurrences in considerable number. However, no other method was suggested to take the place of gastro-enterostomy. Experiments have been made on animals to determine the value of the operation and it has been found (Kelling, Cannon, and Blake) that in a patent pylorus the larger amount of stomach contents goes through the pylorus and not through the anastomotic opening, no matter whether the opening is in the antrum or in the fundus. It has also been noted that there is scarcely any appreciable difference in the emptying time. This has been verified by repeated X-ray examinations of patients who have had gastro-enterostomy—as before, the stomach contents passes through the pylorus. For this reason it has been taken for granted that the gastro-enterostomy has become closed. However, Schnitzler has shown that a correctly performed gastro-enterostomy cannot close, as the opening is completely covered with mucosa; that is, the opening can only become narrow when the technique is incorrectly carried out—for instance, the opening may be made too small or the mucosa may not be painstakingly sutured. In fact one frequently finds, on opening up a patient on whom a gastro-enterostomy has been done, that the old gastrojejunal fistula is entirely patent although the pylorus offers no hindrance. Technical failures that appear after months or years should be excluded as they could have been felt a short time after operation.

After all, more stress should be placed upon the unfinished methods. Gastro-enterostomy is performed with the idea of deflecting from the ulcerated area the pepsin and acid-containing stomach contents. It is not understood just how this is accomplished in the ulcer-free pylorus, for we know that the stomach contents still go through the pylorus and that the mechanical change is produced only when

there is complete pyloric stenosis, in which case the stomach contents must go through the new passage. Therefore, a great many surgeons have decided that gastro-enterostomy does not offer complete cure for ulcer. It has been stated that the change in the chemistry of the contents of the stomach brought about through a gastro-enterostomy does have a healing action as far as the ulcer is concerned. Indeed, through the back current of the alkaline jejunal content, the salt acidity of the stomach is neutralized and so loses its destroying action. This would happen only when the stomach is so completely filled that its contents come in contact with every part of the stomach wall and would be permanent only if the secretion were neutralized. It is true that the stomach contents come in contact only with the part nearest the anastomosis. However, to heal an ulcer by a change in the chemistry it must be more energetic than is possible in the immediate vicinity of the anastomosis. Schnitzler calls attention to the experiments of Pavlow who found that switching the direction of the pulp did not noticeably reduce the secretion in that portion of the stomach. He disregards the fact that there is active stomach secretion by the false lining and that even jejunostomy apparently cannot interfere with the secretory action of the mucosa to such an extent that in every case the ulcer is healed. There must then certainly exist a disproportion between the power of secretion of the stomach glands and that of the intestines with their small irregular supply of alkaline intestinal juice. We know, however, that the internally produced acidation of the stomach produces only subjective relief, as no real clinical or roentgenological cure is effected. Therefore, it is evident that gastro-enterostomy offers only relative rest to an already existing or a future pyloric stenosis and that only in a mechanical way. It can in no way serve as an absolute means of curing a gastroduodenal ulcer. The ulcer still exists and therefore there is the danger of hæmorrhage and perforation, as Schnitzler with the material collected up to 1914 showed. Indeed, the danger is doubled through the appearance of peptic ulcer of the jejunum which requires more skillful handling. Since Braun, in 1899, described the first of such cases, there has appeared the reports of many cases of this dreaded complication following gastro-enterostomy and this has naturally resulted in a loss in popularity of the operation even with those who received so enthusiastically the pyloric deflection as advocated by von Eiselsberg.

It was realized that a more radical means must be found to relieve ulcer of the stomach and duodenum, and resection was decided upon as a more effective means of dealing with the condition. The technique of resection has been perfected to such an extent that at present the method can be depended upon, and the mortality is proportionately low.

In 1914, Schnitzler described the technique of extensive stomach resection for ulcer. By this method only can the secretion of the mucosa be greatly limited. He believes that gastro-enterostomy is indicated only in stenosis of the pylorus with healed ulcer. While radical resection is carried out only in uncomplicated ulcer, one hesitates at times before attempting a palliative operation in perforated ulcer. Apparently one hesitates to begin so difficult a task and prefers to use the old method although it is quite clear that it is not satisfactory. The patient who has but recently recovered from peritonitis due to perforation is placed in a new danger and he still carries the burden of the same difficulty from which he has so long suffered and which has weakened him. How true this is is shown by a case reported by Henry. His patient, in a period of 4 years, was laparotomized for ulcer of the stomach not less than five times, that is for peptic ulcer of the jejunum after gastro-enterostomy. In this case the patient could have been relieved by resection without so many operations. But how many times this has happened, and how many such cases are never reported!

We believe that simple excision of the ulcer is not sufficient, since it does not bring about the most important condition for cure, that is limitation of secretion. If the ulcer has not perforated, resection does give a satisfactory result. (By satisfactory we mean a lasting freedom from recurrence, controlled clinically and roentgenologically.)

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From our experience, we have, therefore, concluded that in perforated ulcer the radical operation is the method of choice if we wish to save the patient from a long-continued illness, for in the end we would be compelled to do the more radical operation, provided the patient has not already succumbed from bleeding or further perforation, and we would then have adhesions to deal with.

The question arises, can resection be done in case the ulcer has perforated? A glance at the case reports will answer the question. We have reported 19 cases, of which 14 were cured and 5 died. All of these cases were admitted to the hospital showing clinically widespread, acute peritonitis.

In all of the cases, the cause of illness was diagnosed, before operation, as perforation of the stomach or duodenum due to an ulcer. In only 4 cases was an iliocæcal incision made, as the history was very hazy and the point of greatest tenderness lay in the cæcal region. It was easy to diagnose perforation because of the quantity of exudate in the abdominal cavity. In these cases two drains were placed through the iliocæcal incision into the pouch of Douglas, and a median incision was made in the epigastric region. In all the cases, perforation was into the abdominal cavity. There was a diffuse, purulent peritonitis, and much gas, exudate, and gastric and duodenal contents, in the abdominal cavity.

In all cases resection of the stomach was done, and more than two-thirds of the stomach was removed, in two cases following the Billroth method and in the rest the Kroenlein-Mikulicz method. The duration of time between perforation and operation was from 1¼ to 36 hours. All patients were given as little anæsthetic as possible, morphine and ether being used. All other details—history, condition of patient, findings at operation, condition after operation, and re-examination—are mentioned in the report of cases. It is noticeable that the ages ranged from 24 to 56 years and that the patients came from all walks of life—which has a bearing on the general state of the patient—nourishment and strength.

It will be noted that the cases in which a good prognosis could be made were treated by non-radical resection, within the first 12 hours after perforation. Up to this time the patients withstand the radical operation very well, provided the operation is carried out under favorable circumstances, by which we mean that the surgeon grasps the situation quickly, employs good technique, and the patient is given but light anæsthesia. Local anæsthesia has no advantage. According to Schnitzler, except for the severe septic condition of the patient, meteorism is the factor most to be dreaded in influencing the result of operation; the presence of gas, pus, or stomach contents is far less important, and large amounts of fibrin or the presence of an œdema of the peritoneum are less influential in the result than is meteorism. In this connection we would call attention to Cases 4, 15, 16, 18, and 19. All these patients were of an unhealthy appearance, breathing was superficial, and pulse was poor. On opening the abdomen a large amount of gas and stomach contents was found, but there was very little meteorism. Extreme flatulency of the intestine contra-indicated the more radical operation, so we used one of the aforementioned methods (not generally used by us). The severe cases (2, 3, 5, and 8) are not exceptions to our views, for in Cases 2, 3, and 5 we were forced to resect despite the bad condition of the patients, due to the poor state of the infiltrated peritoneum about the ulcer. The sutures all cut through so that it was impossible to complete the simple suturing. One covering of omentum and gall bladder offered such poor results that we hesitated to leave the patient with perforation so insecurely covered as perforation would undoubtedly result, and prove fatal. A gastric plication with a connecting gastro-enterostomy, which under favorable circumstances we have tried, would have taken less time than the resection which we carried out. Despite the presence of a high grade meteorism, as found in Cases 5 and 11, the time elapsed since operation argued against resection, still the patients bore the operation well.

Case 8 died of an unusual complication—thrombosis of the subclavian artery with subsequent gangrene of the upper extremity—after having fully recovered from the periton-

itis. The intestinal function was fully restored on the second day after operation, the patient did not vomit, he could even take fluid diet, and he had no pain whatever in the abdomen.

Case 14 was operated upon immediately after perforation and is the only case in which the radical method can be said to have failed. At any rate before operation the patient showed symptoms of immediate collapse and had a rapid pulse; on the other hand the light grade meteorism and the good condition of the intestinal mucosa argued strongly for resection. A beginning pneumonia did not contra-indicate resection as in anæsthetizing the patient only 4 grams of Billroth mixture and 20 grams of ether were used.

A few words regarding technique of operation. The stomach is washed out, the patient anæsthetized with morphine and ether, and the abdomen opened through a median incision from sternum to navel. Even in the most difficult case it has not been necessary to enlarge the original incision. On cutting through the peritoneum, gas under pressure and ordinary fluid contents pour out through the duodenal perforation—infected peritoneal exudate mixed with stomach contents, food remnants, and bile. The stomach is pulled forward, while at the same time the liver is pulled back and upward. Generally at this point everything is visible. In most cases the perforation is located anteriorly in the duodenum or stomach near the pylorus. If the perforation is not found here, by blunt dissection one works backward through the gastrocolic ligament and thus easily reaches the opening through which stomach and intestinal contents are exuding. The perforation is closed temporarily with silk sutures, from pylorus to one-third the way to the cardiac orifice, and the gastrocolic ligament is separated by interrupted ligatures. In this way the view is unobstructed. There is a disadvantage in ligating the ligament in case a resection is to be done in that the stomach, as is known, has a rich blood supply. Our method of resection does not differ from that used by others. We emphasize particularly the necessity of isolating the abdominal field from the rest of the field of operation and especially of carefully sponging away pus and food contents followed by rinsing with hot physiological salt solution. With the exception of Case 5, drainage was not used and the wounds were closed primarily.

In the postoperative treatment we pay special attention to peritonitis. We stimulate the heart function with tonics and overcome the loss of blood by Murphy drips and subcutaneous infusions of physiological salt solution. In severe cases we depend upon intravenous infusion of hypertonic salt solution (15:1,000). In case of vomiting we empty the stomach by inserting carefully a stomach tube, which gives great relief and, so far as we have observed, does no harm. Twenty-four hours after operation the patient receives fluid in the form of tea and water, a teaspoon every half hour. This is increased from day to day. We generally begin giving fluid nourishment *per os* on the fifth or sixth day. On the ninth or tenth day the patient is given semisolids, and 2 weeks after operation the patient is permitted to eat everything, but not too much at a time.

To study our results, we examine our patients, after a lapse of time, with the X-ray (Drs. Eisler and Werner). We have found that no change has taken place in the wall which would indicate ulcer in any of our cases. In Case 4, 1½ years after operation, the patient complained of slight pain at the point of resection; he had never vomited and the pain had no relation to meals. Roentgen examination showed no changes in the wall and the stomach emptied through the anastomosis normally and in the usual time. One patient could not be reached by mail as he had moved, leaving no address. Two other persons could not appear personally, so sent us cards telling us of their condition, which tallied with the above satisfactory findings.

The patients are all free from distress, have good appetites, and eat anything. In all there has been an increase in weight and all have been able to resume their original occupations. They have been cured of the severe suppurating peritonitis, they have had no attacks of severe distress, are free from the fear of a second dangerous operation, and are restored to perfect health.

On the strength of our experience, we would recommend that, wherever possible, stomach resection be done in cases of ulcer of the stomach and duodenum, even in the presence of perforation and peritonitis, also that the older methods be used only when no other method is possible. We have shown that the radical method is practical both as far as operating is concerned and as to result, since with proper indications there is only a small mortality.

CASE 1. Servant, age 29, admitted to hospital, July 11, 1919, with a history of having suffered from stomach trouble for the past 2 years. On walking the patient complains of violent pain located in the epigastrium. On admission temperature was 36.7° C., pulse 96. The facies were hippocratic. Patient was a vigorous man of middle size. Breathing was costal, superficial. He had hiccough, did not vomit, and the abdomen was very tense. Meteorism was present in slight degree. Operation was done 5 hours after perforation. The perforation was 1 centimeter in front of the pylorus, on the anterior gastric wall, close to the lesser curvature. There was a large amount of mucous exudate and of stomach contents and gas in the abdominal cavity. Light meteorism was present in the intestines. There were deposits of fibrin in the serosa which was red and somewhat œdematous. A resection following the Kroenlein method, with primary closure without drainage, was done. Narcotic used: 15 grams mixture, 200 grams ether. Patient had smooth convalescence and was dismissed from the hospital August 15, 1919. We have been unable to locate him with follow-up letter.

CASE 2. Clerk, age 45, admitted September 5, 1919. For 3 weeks patient had complained of distress in the stomach. Last night the pain located in the epigastrium became very severe and he vomited. On admission pulse was 78, temperature 38.3° C. The entire abdomen was tense, hard as a board, and very painful to pressure, chiefly in the epigastric region. Operation was done 27 hours after perforation. Some gas and exudate were found in the abdominal cavity. The perforation was located on the anterior duodenal wall 2 centimeters from the pylorus. Meteorism was quite marked. The serosa was thickened, intensely red, and œdematous, and contained many deposits of fibrin. A resection of the stomach, following the Kroenlein technique, was done, and the wound closed without drainage. Narcotic used: 20 grams mixture, 200 grams ether. Convalescence was smooth and the patient was dismissed as cured on September 20, 1919. The patient writes that he has no trouble at all with his stomach and can eat anything. He has gained 7 kilograms in weight since operation and is perfectly able to continue his usual occupation.

CASE 3. Director, age 52, admitted October 15, 1919. For 16 years he has suffered with stomach trouble which has been severe at times and at times he has been free from distress. Twelve years ago he had bloody stools. While walking near the hospital, perforation occurred. On admission the pulse was 90, temperature 37.5°C. Patient is a vigorous, well nourished man. The abdomen is hard as a board and is sensitive to pressure, chiefly in the epigastric region. He has a violent hiccough. Operation was done an hour and a half after perforation. Some gas and stomach contents were present in the abdominal cavity. The perforation was located in the mid portion of the pyloric wall, and produced a contact ulcer back of it. No meteorism was present. The serosa was red, not thickened, and contained no deposits of fibrin. A resection of the stomach was done, following the Kroenlein technique. The wound was closed without drainage. Narcotic used: 20 grams mixture, 300 grams ether. Convalescence was smooth and patient was dismissed as cured, October 25, 1919. On July 15, 1921, patient states that he feels perfectly well, can eat anything, and in considerably greater quantity than formerly. He has gained 6 kilograms in weight and is perfectly capable of continuing with his work. Roentgen examination shows a well contracted stump on the left side and emptying time is prompt. No change in the walls is to be found. No point is painful on pressure.

CASE 4. Patient, woman, laborer, age 35, admitted November 11, 1919. For 4 months patient has suffered with distress in stomach and pain in the back. She has vomited. Perforation occurred on day of admission and was associated with extreme pain. On admission pulse was 120, arhythmic; temperature 36.4°C. Patient was prostrated and badly emaciated. Breathing was superficial, and the cheeks were pale and sunken. The abdomen was inflated, and extremely painful to pressure. She hiccoughed. Operation was done 11 hours after operation. Much gas and exudate with stomach contents were found in the abdominal cavity. There was a perforation near the lesser curvature in the pyloric third. Inside the stomach there were 6 ulcers. There was light meteorism, the serosa was intensely red, greatly thickened, œdematous, and contained deposits of fibrin. Narcotic used: 15 grams mixture, 200 grams ether. Convalescence was smooth and patient was dismissed as cured on January 23, 1920. Up to July, 1921, patient remained entirely free of complaint. Since that time she has had two attacks of pain without eructation and vomiting, not associated with eating. She can eat anything, has gained 4 kilograms in weight. Roentgen examination shows stump of moderate size, which empties very quickly. The jejunum is somewhat dilated. No change in the walls is to be found.

CASE 5. Woman, peddler, age 56, entered hospital, November 29, 1919. For some time past she has been suffering from gall stones. On the day before admission to hospital she had violent pain in the epigastrium. She did not vomit. She had crural pains. Bowels had not moved for 2 days. Exam-

ination shows pulse 120, arhythmic; temperature 37.6° C. Patient is woman of moderate strength with hippocratic facies. The abdomen is very tense and painful on pressure, chiefly in the iliocæcal region. She vomited profusely and had violent hiccough. Operation was done 36 hours after perforation. Enormous quantities of gas and pus with stomach contents were found in the abdominal cavity. The serosa was thickened, intensely red, and contained deposits of fibrin. The perforation was located on the anterior wall of the stomach close to the pylorus. A resection of the stomach, after the method of Kroenlein, was done and two drains were placed in the iliocæcal incision. Narcotic used: 10 grams mixture, 200 grams ether. Patient died December 3, 1919. Postmortem examination showed a subacute tuberculosis of both superior lobes, brown atrophy of the heart, and a diffuse purulent peritonitis.

CASE 6. Printer, age 51 years, was admitted November 29, 1919. For 30 years he had suffered periodic distress in the stomach. On day of admission patient suffered a sudden, acute attack of extreme pain in the epigastric region, associated with violent vomiting. Pulse was 96, temperature 36.7°C. Patient was of moderate strength, and appeared ill. The abdomen was drawn in and tense, hard as a board. There was a tympanitic sound underneath the sternum. Time elapsed between perforation and operation not known. Operation revealed much gas and some exudate in the abdominal cavity. The perforation was located on the anterior pyloric wall. There was a light meteorism. The serosa was red, somewhat thickened, and contained deposits of fibrin. A stomach resection after the method of Kroenlein was done and the wound closed without drainage. Narcotic used: 10 grams mixture, 80 grams ether. Convalescence was smooth and patient was dismissed as cured on February 9, 1920. Patient gained 7 kilograms in weight, can eat anything, and has a good appetite. He has returned to his work as printer. On July 15, 1921, roentgenogram showed a well contracted stump of moderate size, emptying time short, and no change in walls. At this time there is no point which is painful on pressure.

CASE 7. Grinder, age 29, was admitted February 26, 1920. For 9 years patient has suffered from stomach trouble, with intervals of 5 to 6 weeks in which he would be free of distress. Perforation occurred with intense pain after patient had eaten of a rich meal. There was no vomiting and no hiccough. On admission the pulse was 60, temperature 37.2° C. Patient is a vigorous man with sunken cheeks. He has a diffuse bronchitis. The abdominal walls are hard as a board. The epigastric region is extremely sensitive. Operation was done 5 hours after perforation. Much gas and some free exudate were found in the abdominal cavity, chiefly beneath the liver. Perforation was located on the duodenum, close to the pylorus. No meteorism was present. The serosa was red, somewhat œdematous. A stomach resection after the method of Kroenlein was done and the abdominal walls sutured. Narcotics used: 5 grams mixture, 110 grams ether. Convalescence was smooth, and the patient was dismissed as cured on March 28, 1921. The patient gained 4 kilograms in weight, eats well and anything—has no trouble at all.

CASE 8. Ironworker, age 29, admitted March 15, 1920. For 3 years patient had suffered with stomach trouble. Perforation was associated with intense pain so that patient almost fainted. On admission pulse was 72, temperature 36.7° C. Patient was a vigorous man with sunken cheeks. Breathing was short and superficial. The abdomen was tense and hard as a board. Sensitiveness was evident, chiefly in the epigastric region. He had violent hiccough. Operation was done 4¾ hours after perforation. On opening the abdomen a large amount of gas and gastric juice was found. Perforation was located on the anterior wall of the duodenum close to the pylorus. There was no meteorism. The serosa was only slightly thickened and red. Stomach resection after the method of Kroenlein was done and the abdominal wound closed. Narcotic used: 18 grams mixture, 110 grams ether. Convalescence was smooth and the patient was dismissed as cured on March 28, 1920. On July 13, 1921, the patient reports that he is free from distress, can eat anything and in larger quantities than before operation. Roentgenological examination shows a well contracted stump of moderate size, with prompt emptying of the stomach contents. There is no change evident in the walls and no point of sensitiveness on pressure.

CASE 9. Tailor, age 24, entered hospital June 13, 1920. Until 2 days ago he had never had trouble with his stomach, but at this time he has severe pain and on the afternoon of the day before entering hospital he was seized suddenly with terrific pain in the epigastrium. He did not hicccough and did not vomit. On admission the pulse was 90, temperature 36.3° C. He was a moderately strong man, slightly cyanotic. The region of the epigastrium was tense and extremely sensitive to pressure. Sixteen and one-half hours elapsed between perforation and operation. On opening the abdominal cavity there were found much gas, exudate, and gastric contents. Perforation was located on the anterior wall of the duodenum, close to the pylorus. There was present a light meteorism, and the serosa was intensely red, much thickened, and contained deposits of fibrin. Stomach resection after the Kroenlein method was done and the abdominal wound closed without drainage. Narcotic used: 5 grams mixture, 180 grams ether. Convalescence was interrupted by a bilateral pleurisy. Patient was dismissed cured on July 13, 1920. Aside from a short period, November 19, to December 17, 1920, when an operation for abdominal pressure was done, patient has had no trouble at all. He can eat anything and has gained 4 kilograms in weight. X-ray examination shows a well contracted resection stump of moderate

size. Emptying time is moderately prompt. There is no change in the walls, and no point painful to pressure.

CASE 10. Director, entered hospital, March 3, 1920. For a year patient has been suffering from stomach trouble. Perforation was brought on by lifting a heavy load. On admission pulse was 85, temperature 37.5° C. Patient is weak with hippocratic facies. The abdomen is hard as a board, and is especially sensitive to pressure in the epigastric region. Operation was done 7 hours after perforation. On opening the abdominal cavity much gas and exudate were found. The perforation was situated on the anterior wall of the duodenum, close to the pylorus. There was present a light meteorism, the serosa was red, thick, and there were fibrin deposits on the serosa. Stomach resection was done following the method of Kroenlein and the abdominal wound closed. Narcotic used: 20 grams mixture, 220 grams ether. Convalescence was smooth and the patient was dismissed as cured on April 30, 1920. On July 15, 1921, patient reports that he is feeling perfectly well, can eat anything, and is able to continue with his usual occupation. He has gained 4 kilograms in weight. X-ray examination shows a well contracted resection stump of moderate size, and emptying time is moderately prompt. There is no change in the walls and no point painful on pressure.

CASE 11. Workman, age 50, entered hospital September 10, 1920. For 1 year he has been suffering with stomach trouble. After a heavy meal of cabbage, he was suddenly seized with intense pain in the epigastrium, and violent hiccough. Examination shows pulse 136, temperature 37.3° C. Patient is weak and ill looking. The abdomen is drawn in and is extremely sensitive to pressure. Operation was done 18 hours after perforation. On opening the abdomen large quantities of gas and stomach contents were found. The perforation was located on the anterior pyloric wall. There was marked meteorism. The serosa was thickened, red, and on it were deposits of fibrin. It was easily perforated. A stomach resection after the method of Kroenlein was done and the abdominal wound closed. Narcotics used: 100 grams ether. The patient died September 11, 1920. Postmortem examination showed diffuse peritonitis and weakness of the heart.

CASE 12. Merchant, age 41, entered hospital September 15, 1920. Patient had formerly been well until 20 hours ago when he was seized with intense pain in the region of the stomach. Soon afterward he had violent colic-like pain throughout the abdomen, and hiccoughed violently. On admission pulse was 130, temperature 38.7° C. Patient was a vigorous man who showed the evidences of being very ill. He vomited profusely. The abdomen was distended and very painful to pressure. Patient was operated upon 20 hours after perforation. On opening the abdomen much gas and rich exudate were found. The perforation was found on the anterior duodenal wall, close to the pylorus. Strong meteorism was present. The serosa was red, contained many deposits of fibrin, and was easily perforated. A stomach resection after the Kroenlein method was done and the abdominal wound closed. Narcotic used: 4 grams mixture, 100 grams ether. Patient died September 17, 1920. Postmortem examination showed a right-sided hydronephrosis with stenosis of the right ureter, directly in front of the urinary bladder, also diffuse peritonitis.

CASE 13. Workman, age 24, entered hospital October 7, 1920. For 8 days he has had pain in the stomach, chiefly at night. Perforation occurred during violent pain and vomiting. On admission pulse was 96, temperature 37.8° C. Patient is a vigorous man showing evidence of extreme illness. The abdomen is intensely sensitive to pressure, tense, and as hard as a board. Patient hiccoughed violently. Operation was done 11 hours after perforation. On opening the abdominal cavity some gas and exudate were found. The perforation was found on the anterior duodenal wall, close to the pylorus. There was light meteorism. The serosa was thickened and reddened. Stomach resection after the Kroenlein method was done and the abdominal wound closed. Narcotic used: 4 grams mixture, 100 grams ether. Convalescence was smooth and patient was dismissed as cured on November 28, 1920, after being treated with arsenic. Report on July 28, 1921, states that the patient is feeling perfectly well, can digest all food, and has increased 7 kilograms in weight. X-ray examination shows resection stump on left side and rather prompt emptying time. There is no change in the walls. Somewhat to the right of the stomach is a point painful to pressure.

CASE 14. Workman, age 42, entered hospital October 28, 1920. For 4 years patient has suffered with severe distress of the stomach. Roentgen examination showed an ulcer of the stomach. Perforation followed a period free from pain and was associated with intense pain and vomiting. On admission pulse was 130, temperature 36.4° C. Patient is a vigorous man, with peritonitic facies. The abdomen is distended, tense, and hard as a board. Operation was done 4 hours after perforation. On opening the abdominal cavity much gas and some stomach contents were found. The perforation was found on the lesser curvature near the pylorus. There was some meteorism. The serosa was red, somewhat thickened. Stomach resection after the method of Kroenlein was done and the abdominal wound closed. Narcotic used: 4 grams mixture, 140 grams ether. Patient died October 29, 1920. Postmortem showed lobular pneumonia and diffuse peritonitis.

CASE 15. Workman, age 49, entered hospital December 22, 1920. For 18 years patient has suffered with stomach trouble. Pain comes on 1 hour after eating and also at night, and he suffers with eructations. Perforation was associated with intense pain but patient did not vomit. On admission pulse was 104, temperature 36.3° C. Patient is weak and shows evidence of his illness. The abdomen is

drawn in, and intensely painful to pressure, especially in the epigastrium. Operation was done 5 hours after perforation. On opening the abdominal cavity a large amount of gas and stomach contents was found. The perforation was found on the lesser curvature near the pylorus. There is some meteorism. The peritoneum is red and œdematous. A stomach resection after the method of Kroenlein was done and the abdominal wound closed. Narcotic used: 2 grams mixture, 280 grams ether. Convalescence was smooth and the patient was dismissed as cured on January 5, 1921. On June 12, 1921, patient reports no trouble at all, he can eat anything, and has gained 8 kilograms in weight. X-ray examination shows a resection on the left side and prompt emptying time. There is no change in the walls and no point painful to pressure.

CASE 16. Servant, age 46, entered hospital December 29, 1920. For 14 years patient has suffered with stomach distress—severe colic and cramps in the epigastric region, pain extending from the sacrum to the shoulders, sour eructation. For 10 days the pain has increased in intensity and on the evening before entering hospital patient experienced feeling as though something gave way in abdomen. On admission pulse was 160, temperature 36.6° C. The patient looks miserable, the abdomen is drawn in and is hard as a board. He vomits violently. Operation was done 6 hours after perforation. On opening the abdomen much gas and exudate was found. The perforation was located on the lesser curvature close to the pylorus. There is light meteorism. The peritoneum is red and œdematous. A stomach resection following the method of Kroenlein was done and the abdominal wound closed. Narcotic used: 4 grams mixture, 110 grams ether. Convalescence was smooth and patient was dismissed as cured on February 2, 1921. Letter from patient in middle of June, 1921, states that he is entirely free of distress.

CASE 17. Coachman, age 44, entered hospital April 19, 1921. For 4 weeks he has had stomach trouble, and on evening before entering hospital, while lifting a heavy load, patient was seized with violent pain, and perforation occurred. Examination on admission showed pulse 120, temperature 37° C. Patient is a vigorous man but is extremely dyspnœic. The legs are drawn to the body, the abdomen is hard as a board and is sensitive to pressure chiefly in the gastric region. Patient hiccoughs. Operation was done 13 hours after perforation. On opening the abdominal cavity a large quantity of gas and exudate was found. The perforation was found on the anterior duodenal wall, near the pylorus. There was light meteorism. The peritoneum was red, thickened, œdematous, and showed deposits of fibrin. A stomach resection after the Kroenlein method was done and the wound closed. Narcotic used: 28 grams mixture and 100 grams ether. Patient died April 23, 1921. Postmortem examination showed thrombosis of left subclavian artery with gangrene of the left upper extremity.

CASE 18. Workman, age 34, admitted April 14, 1920. For 1 year patient has suffered with stomach trouble. Perforation was associated with intense pain and no vomiting. On admission pulse was 120 and weak, temperature 36.3° C. Patient was a vigorous man but showed evidence of illness. The abdomen was tense, somewhat inflated, painful on pressure, chiefly in the umbilical region. Operation was done 5 hours after perforation. On opening the abdominal cavity a large amount of gas and exudate was found. The perforation was on the posterior duodenal wall, near the pylorus. There was light meteorism. The peritoneum was red, somewhat œdematous. A stomach resection after the method of Kroenlein was done and the abdominal wound closed. Narcotic used: 40 grams mixture, 240 grams ether. Convalescence was smooth and patient was dismissed as cured on May 2, 1921. On July 12, 1921, patient is entirely free of distress, can eat anything and in larger quantities, and is able to continue with his usual occupation. X-ray examination shows a short, somewhat dilated stump, with prompt emptying time. There is no change in the walls. There is a point painful to pressure just outside and to the right of the stump.

CASE 19. Machinist, age 46, entered hospital May 31, 1920. Patient had suffered periodic distress in stomach for past 20 years. He had severe attacks of vomiting of sour masses. Perforation occurred when patient was bending during an attack of pain and vomiting. On admission pulse was 96, temperature 37.6° C. Patient is of moderate strength, breathing is superficial, and the cheeks sunken. The abdomen is somewhat distended and is painful to pressure throughout. Operation was done 8 hours after perforation. On opening the abdominal cavity a large quantity of gas and stomach contents was found. The perforation was located on the anterior duodenal wall, close to the pylorus. The peritoneum was red and a little thickened. A stomach resection after the Billroth I method was done and the wound closed. Narcotic used: 15 grams mixture, 80 grams ether. Convalescence was smooth and patient was dismissed as cured on June 11, 1921. Report on July 12, 1921 shows patient entirely free of complaint. He eats four good meals a day and has gained 5 kilograms in weight. X-ray examination shows a moderate sized stump with moderate emptying time. There is no change in the walls and no point painful on pressure.

LITERATURE

KAUSCH. Handbuch der Chirurgie. Von Bruns, Garre, and Kuettner. vol. iii.
HABERER. Wien. klin. Wchnschr., 1919, No. 16.
EUNICKE. Deutsche med. Wchnschr., 1919, No. 28.
MASSARI. Wien. klin. Wchnschr., 1920, No. 12.
SCHWARZMANN. Wien. klin. Wchnschr., 1920, No. 22.
HENRY. Recurrent gastric perforations. Surg., Gynec. & Obst., 1921, xxxii.
KELLING. Beitr. z. klin. Chir., 1908, lvii.
CANNON and BLAKE. Ann. Surg., 1904, xli.
SCHNITZLER. Med. Klin., 1914, Nos. 16 and 17.

MARGINAL ULCER AFTER A MODIFIED PÓLYA OPERATION

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THE question of marginal or gastro-jejunal ulcer following a gastro-enterostomy has, of late, interested many surgeons and a large number of such cases has been reported in recent years. It would seem from reading these reports that a gastro-enterostomy for a gastric or duodenal ulcer is a poor operation, and yet, for the majority of cases that one sees, it is apparently the best operative proceeding that we have to offer, at present. The one operation that can possibly replace a gastro-enterostomy is the pyloroplasty of Horsley, and the field for the latter is limited to a comparatively small number of duodenal or postpyloric ulcers without extensive cicatrization and very limited in size. A marginal ulcer is known to have followed other anastomosing operations as Erdmann has described one after a Billroth No. 2, and we are now describing one after a modified Pólya operation.

As far as we know, at present, the cause of gastric and duodenal ulcer is probably a hæmatogenous infection from some other primary focus, as demonstrated by Rosenow, by some specific bacterium, and it would seem quite possible that the cause of a marginal ulcer in a patient after a gastro-enterostomy is a reinfection by the same bacteria on account of non-removal of the original primary focus. It is very possible that there are other factors which aid in the formation of marginal ulcers and yet these may be purely secondary. Among them we may mention the following:

Non-absorbable suture material. It is true that silk and linen thread have been found in many marginal ulcers and yet many cases have been reported where only absorbable suture material had been used throughout the operation.

The use of heavy clamps. Trauma and lacerations from the use of Roosevelt, Allis, and other tightly applied clamps may and may not help in the formation of these ulcers. They are not reported after the large resections for carcinoma of the stomach in which traumatism to the stomach wall is of necessity much greater and in which heavier clamps are often applied with greater force. It is possible, however, that marginal ulcers may occur after these resections and may be obscured by local recurrences. The fact remains, however, that marginal ulcers are reported after operations by surgeons who use no clamps whatsoever.

Syphilis. Erdmann mentions this disease as having been shown in three out of five ulcers excised and examined microscopically, and yet this condition can not be given as a definite causative factor in the majority of cases.

Chemical changes. Hyperacidity has often been blamed for the formation of gastric and duodenal ulcers and at present, according to Horsley, it is the most important etiological factor in the formation of marginal ulcers. And yet one is forced to wonder whether too much importance has not been laid on this point. It has never been definitely decided whether hyperchlorhydria is a causative factor of ulcer or whether it is only found when the ulcer has actually made its appearance. Cases are frequently seen in which there is no excessive secretion of hydrochloric acid.

It is true that the marginal ulcer is usually seen in the wall of the stoma itself or in the wall of the jejunum opposite the stoma, a fact which would seem to show that the ulcer is due to the chemical irritation of the acid gastric juice not neutralized by the alkaline duodenal contents. Yet we know that many ulcer cases are seen where there is no hyperacidity, and Lewisohn has lately reported a case of marginal ulcer where a hypoacidity was present in the gastric contents.

Several authors have used the words "personal idiosyncrasy in the production or reproduction of marginal ulcers." On analysis, this simply means that an operation was done for the cure of an ulcer and that, in spite of dietary and medical care, another ulcer ap-

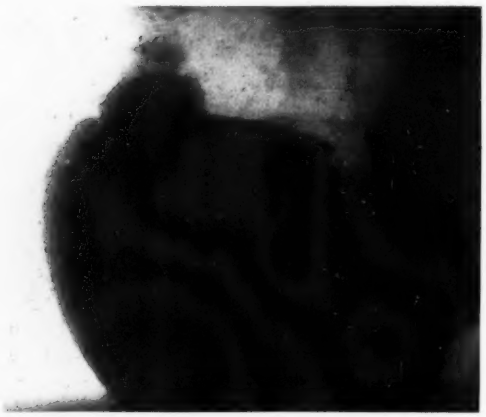

Fig. 1. Before operation. Marked pyloric stenosis; pyloric end of stomach greatly dilated; cap distorted beyond recognition; pyloric sphincter deformed.

peared at a new site, although the original one had been, apparently, cured in the meantime. In two of Erdmann's cases there were, apparently, two new ulcers appearing since the operation for the original one. In all probability, "idiosyncracy" simply means reinfection at a new site from an undiscovered primary focus. Where such a primary focus is discovered and removed, medical and dietary treatment probably will cure many ulcers. A gastro-enterostomy, without the removal of the primary focus, will probably permanently cure many ulcers where the agent of infection from the primary focus is one of slight virulence. Where, however, the virulence of the bacteria is great, a gastro-enterostomy may cure the original ulcer but a new ulcer may be caused by the same bacteria from the unremoved, primary focus, in the new stoma which is an area, irritated by non-absorbable sutures, traumatized by heavy clamps, or burnt by highly acid gastric contents, and, therefore, an area the resistance of which to bacterial infection has been materially decreased.

The following case is reported because, in a search through the literature, no other case of a marginal ulcer following a Pólya operation was found and because the clinical course of the formation of the second ulcer could be followed so closely.

The patient was a very energetic white man of 45, the editor of a scientific journal whose work required long hours and who, for many years, had had his meals at irregular intervals. He entered the French Hospital on July 7, 1916, with the following history: He had had the usual diseases of childhood and typhoid at the age of 22. Otherwise he had been well until 5 years before when he began to have attacks of indigestion which usually came on after a period of hard work. These attacks were characterized by some nausea, acid eructations, a feeling of fullness in the epigastrium, but no real pain. The attacks lasted for only a few hours at a time. In the early spring of 1916 he could not eat a real meal without vomiting and so lived by taking small quantities of food at frequent intervals. There had never been any blood in the vomitus or in the stools. His bowels had been regular up to within a few months before his admission to the hospital when he became very constipated. He had lost 20 to 25 pounds in weight in the last 2 years. Physical examination at the time of his admission showed nothing abnormal except for a moderate amount of gastric dilatation. He had a red cell count of 4,700,000, a white count of 15,400 and a hæmoglobin of 90 per cent. Gastric analysis showed a total acidity of 105, and a free hydrochloric acid content of 65, and no blood. The fæces were negative for blood. The examination of the urine was negative as was the Wassermann. The roentgenographic findings by Dr. Cole, on June 15, 1916 were as follows:

The stomach was dilated and prolapsed. The peristalsis was very active, of the four-cycle type; equal on the greater and lesser curvatures and unobstructed. The jejunum showed distinctly. The duodenum passed down behind the pyloric end of the stomach. The cap was distorted almost beyond recognition and the sphincter was almost obliterated. The pyloric end of the stomach expanded and contracted normally except for the extreme gastric dilatation. At 2 hours the stomach showed that relatively little food had passed out of the stomach and at 6 hours there was still a great deal of retention in the stomach.

From these plates Dr. Cole made a positive diagnosis of a postpyloric ulce with marked obstruction.

He was operated on July 9, 1916, by Dr. Hoguet, the following operation being done. The abdomen was opened by a right rectus incision 4 inches in length. The stomach was found greatly dilated and a broad band of scar tissue was present just to the left of the pyloric ring. There was a broad, indurated ulcer, mostly on the anterior surface, immediately to the right of the pyloric ring and extending outward on the duodenum. The stomach was completely divided from the lesser to the greater curvature about 2 inches to the left of the pylorus. A number of adhesions were found when the posterior stomach wall was divided, and these were tied and cut. The duodenal stump was then closed with a through-and-through suture of No. 1 chromic gut and the suture line inverted by a continuous Lembert

Fig. 2. After operation. No pyloric stenosis; stomach normal in size and shape; no cap shown; normal gastric peristalsis.

Fig. 3. First recurrence after operation; stenosis at stoma; stomach dilated; no cap; arrow indicates crater of ulcer of jejunum; hyperperistalsis.

suture of Pagenstecher. An end-to-side anastomosis was made between the cut end of the stomach and the jejunum, a Pagenstecher suture being used for the serosal stitch and No. 1 chromic gut for the mucosa. The jejunum lateral to the anastomosis was then sutured to the sides of the hole in the gastrocolic omentum. The abdominal wall was closed by layers in the usual way.

The patient made an unusually good recovery from this operation and was discharged from the hospital on July 26, 1916, under medical and dietary supervision.

A roentgenographic examination made in December, 1916, showed: the size and shape of the stomach and also its position to be almost perfect. From the plates one could not tell that there had been any operation on the stomach itself. The 2 hour plates showed the stomach almost completely empty, there being only a slight trace of barium in the stomach at that time. The 6 hour plate showed the stomach completely empty, the head of the column in the ascending colon and the tail in the coils of the ileum.

The patient resumed his work in the fall of 1916 and complained of nothing until February, 1917, when he began to notice some acid eructations, which were followed in a few days by nausea and vomiting. The vomiting had no relation to his meals but usually occurred at night and consisted generally of liquids without any undigested food material and never contained blood. He had no abdominal pain.

A roentgenographic series made March 13, 1917, showed the stomach greatly dilated and somewhat prolapsed. The peristalsis was hyperactive and yet little food had entered the jejunum. At 2 hours there was shown marked retention and at 6 hours there was the same retention as at 2 hours. Just beyond the stoma in the jejunum there was evidence of a definite crater about one-half inch in length and one-fourth inch in width. Surrounding this was an area which seemed to be a large indurated mass constricting the lumen of the gut at that point. From these plates Dr. Cole made a positive diagnosis of a jejunal ulcer with a considerable amount of induration, causing obstruction of the passage of food.

He entered the hospital in the latter part of February, 1917, and was put on a Lenhartz diet and kept in bed. His symptoms rapidly disappeared, and he was discharged on March 25, 1917. He remained at home for several weeks under treatment. He was perfectly well except for a slight attack of indigestion in December, 1918, which only lasted a few weeks and which disappeared rapidly under dietary treatment. Until the fall of 1920 the patient was free from symptoms but in November began to complain of a feeling of general malaise and an uneasy feeling in the stomach. On December 13, 1920, he had, for the first time, a definite attack of pain in the epigastrium which came on a few hours after a meal and was somewhat relieved by bicarbonate of soda. The pain kept up for several days and was accompanied by some nausea. There was no blood in the stools. He was readmitted to the hospital December 20, 1920. Physical examination at that time showed slight enlargement of the stomach and a very slight tenderness in the mid-line just above the umbilicus. A roentgenographic examination at this time did not show the duodenum and the cap. The stoma showed distinctly and there was a persistent fleck of barium just above it. There was a marked gastric retention at 6 hours, which persisted up to 24 hours. There was also a retention of some barium in the ascending colon in the 24-hour pictures.

From this examination Dr. Cole made a positive diagnosis of a rather large ulcer on the upper aspect of the jejunum just beyond the gastric stoma with

Fig. 4. Second recurrence after operation. Stenosis of stoma; stomach dilated; no cap; arrow indicates crater of ulcer of jejunum; hyperperistalsis.

Fig. 5. Second recurrence just before final operation. Stenosis of stomach; arrow indicates small crater surrounded by much induration. Hyperperistalsis.

enough induration to cause obstruction and 24-hour retention.

He remained in the hospital until January 10, 1921, under strict dietary treatment and then returned to his home, where the medical treatment was continued under the supervision of Dr. M. A. Ramirez. A roentgenographic examination was made in March, 1921.

A series of plates made of the stomach immediately after a bismuth meal was administered, showed very considerable dilatation of the stomach and excessive peristalsis. In spite of the peristalsis there was definite evidence of stenosis at the point of the gastric stoma which was contracted to about the size of a goose quill. There was definite evidence of a crater just beyond the stomach and evidence of dilatation of the jejunum just proximal and just distal to the stoma. There was an immense amount of induration surrounding the stoma, particularly above it. This finding, compared with the previous examination, showed practically the same condition with the induration and cicatrical contraction greater at this time.

There was very definite evidence of gastric retention up to 24 hours.

From a study of these plates Dr. Cole made a diagnosis of a jejunal ulcer just beyond the stoma, with marked contraction of the stomach, and surrounded by a large amount of induration.

After a consultation it was decided to operate for the marginal ulcer. The patient re-entered the hospital on April 3, 1921, and the following operation was done: On opening the abdomen through a right rectus incision the omentum was found adherent to the under surface of the abdominal wall. The gall bladder was found to be normal, but the appendix showed definite signs of a chronic inflammatory process and was bound down at its center. It was removed in the usual way. The cæcum was then explored, and several strong bands of adhesions running across it were found and divided. These bands were evidently the cause of the stasis in the ascending colon that had been noted in one of the roentgenograms. The anastomosis between stomach and jejunum was found covered by many adhesions. The opening in the gastrocolic omentum was found from below and was dissected away from the stoma. The ulcer was at the margin of the anastomosis and was larger on the gastric than on the jejunal side, but formed a definite ring of fibrous tissue completely surrounding the stoma. Directly opposite the stoma was a pouch in the jejunal wall about three-quarters of an inch deep. The anastomosis was cut directly across with a knife and the opening in the stomach closed with two layers of chromic catgut. The opening in the jejunum was closed at right angles to its long axis with two sutures of chromic. The caliber of the jejunum was then tested and found to be large enough. A posterior gastro-enterostomy was then done about 1.5 inches away from the

sutured end of the stomach, the jejunum being used at about 4 inches distal to the old anastomosis. The abdomen was closed in the usual way.

Several points in the operative findings are worthy of consideration. First, that the ulcer had apparently started in the wall of the jejunum, had enlarged so that it had encircled the stoma and then had cicatrized so that, at operation, the latter was scarcely as large as a lead pencil in its diameter.

The second point is that no remains of non-absorbable suture material were found in dividing the old anastomosis.

The third is that several bands of stout adhesions were found constricting and angulating the ascending colon and an appendix that was distinctly pathological. In this case, might not the inflamed appendix or the constricted colon have been the cause of both the first and second ulcers?

The patient did very well until the sixth day after operation when the nurse reported a discharge on the dressings. When the latter were removed it was found that the wound had opened for about 3 inches at its lower angle, through the peritoneum. This was immediately resutured under local anæsthesia with a number of through-and-through silkworm-gut sutures. The patient did well for 4 days more when his temperature and pulse rate started to rise, and he died on the fourteenth day after the first operation.

A wound inspection was made and showed no general peritoneal infection. The stomach was not distended and the new anastomosis seemed to be firmly healed. There was no kinking near the stoma. There was a mass of omental adhesions over the sutured pyloric end of the stomach and on separating these it was found that the sutured ends of the gastric walls at the site of the old stoma had not united and that a cavity had formed at this point between stomach walls and omentum, in which there was about an ounce of pus. The sutured incision in the wall of the jejunum where the old anastomosis had been divided, was tight.

MEGASIGMOID, MEGARECTUM, FÆCAL BOLUS

By FREDERICK G. CORBIN, M.D., C.M., Mendoza, Argentine Republic

I WISH to use the word megasigmoid as that, to my mind, best implies the pathological condition I am about to describe. For the last 22 years in this city of Mendoza, we have been fighting megarectosigmoid and fæcal bolus, and have been studying its cause and effect. During this time I have observed from an experience with some 200 cases, the following: First, that the world's literature on this subject is almost *nil*, especially in view of the gravity and frequency of this disease here. Second, that many deaths in Mendoza are caused by megasigmoid or are rather direct consequences of this condition. Third, that appendicitis, gastric ulcer, duodenal ulcer, and cancer of the rectum, about which so much appears in the world's literature, all together do not cause so many deaths in Mendoza as do megasigmoid and its direct sequelæ. Fourth, that megasigmoid is much more common in Mendoza than in Europe, North America, or Buenos Aires. Fifth, that there is a great deal of confusion among authors, many describing congenital megacolon and megasigmoid and megarectosigmoid as if they were all one disease.

I do not remember ever seeing or hearing of a case of fæcal tumor in the Montreal General Hospital during my student years at McGill, from 1885 to 1890. The same may be said of my 6 months in 1890 spent in the British Hospital in Buenos Aires. I saw a few cases of coprostasis which, with a simple soap suds enema followed or preceded by a dose of castor oil, were easily relieved.

Of my early years of practice I have no written records, but in the Rio Negro from 1890 to 1894, I remember only one case of fæcal tumor and one of volvulus of the sigmoid; the latter in a police autopsy; poisoning was given as the cause of death.

In Chascomus, where from 1894 to 1898, I did the surgery of both hospitals (80 beds), I remember only one case of fæcal tumor. In 1896 I saw and helped the late Dr. Andres

Megarectosigmoid. Roentgenogram taken after 4,000 gram enema containing 500 grams barium. The megarectosigmoid extends up into the thorax, then the normal (?) sigmoid comes back to the pelvis and becomes the descending colon in normal situation. The cæcum and ascending colon are not seen as it is covered by the megasigmoid.

This case is noteworthy only because it is in a girl of 18 years. Case of Dr. Metraux, January, 1922.

Llobet do his operation: i.e., laparotomy and crushing of the fæcal mass *in situ*. Later on I will again speak of this.

In the Buenos Aires hospitals they tell me that *now* this disease is comparatively rare. There are cases and with the aid of the X-ray they find many megacolons and megasigmoids without fæcal tumor, and strange to say, without the patients suffering any disagreeable consequences. However, 20 years ago I believe it was much more common to see the fæcal tumor as a consequence or possibly as a cause of megasigmoid, more especially here in Mendoza where, as I said before, it was and is much more common than in other sections.

Megacolon is considered by European authors as a congenital disease, and megasigmoid, I believe with my colleagues here, is a consequence of fæcal tumor which is at first caused by simple sigmoid stasis.

We may be mistaken. These enormously dilated sigmoids, with their walls three or four times the normal thickness, may have originally been congenital megacolons which, by the accumulation of excrement, at times in enormous quantities, and the extraordinary muscular forces called into play to expulse it, has produced an entirely new condition in its muscular coat. The condition may be compared with the change produced in the normal individual who takes up physical exercise, gymnasium work, places himself on a diet, in other words "trains" and as a result becomes a giant in strength and endurance; or to the development of the gastrocnemius of the cyclist. In the same way it is possible that the presence of fæcal matter in the lower part of the colon and upper part of the rectum, produces in their muscular coat an excessive development with fibrous changes later on. Is it necessary that there be a congenital dilatation or an abnormal fold in the sigmoid for this to happen? No! At least I believe that all this may and does happen in a normal sigmoid and that it is a frequent occurrence.

In nearly all the cases which I have seen and examined (some 200) I have found on digital examination a narrowing in the rectum which corresponds to the peritoneal fold which exists normally around the first part of the rectum, but only in *front* of the second part; that is to say, in the line of division, according to our anatomical nomenclature, between the first and second part of the rectum (Perthes' fold). Above this fold the fæcal matter becomes lodged and accumulates; below this point it does not pass. Only twice have I seen fæcaloma in the ascending colon and only once in the transverse.

Normally, according to the best authors, the narrowest part of the large intestine is the first part of the rectum. This narrow part, conical in shape, is smallest exactly at the point mentioned above (Perthes' fold). Passing this we come to the wider part which

forms the rectal pouch. This first part of the rectum, at the same time the narrowest, forms an "S" from left to right and from before backward.

In 95 per cent of my cases of fæcal accumulation, this first part of the rectum and the iliac sigmoid immediately above are the situations chosen. *Here* is the dilatation; *here* is the tumor; and *here* is the degeneration of the muscular coat of the intestine. It is here that the fibrous tissue replaces the muscular to a great extent; and the normally elastic intestine loses all its contractile power. Soap suds and oils clean out the rectal pouch in the second part of the rectum; but the fæcaloma, big and hard in the rectosigmoid segment, resists all medical treatment, sometimes for weeks. This mass by its own weight, given a long and weak mesosigmoid, seeks for the place of least resistance, migrating to the middle line and often to the right side of the abdomen.

This emigration of the sigmoid and the first part of the rectum from its normal situation is undoubtedly favored by the mesos mentioned, always long and weak in persons who suffer from this disease. This lengthening may be the *congenital* part in these cases, as Barth claims. There is in front a muscular wall, to the left and behind a bony wall, making the middle line and the right side the natural localities for the final enlargement of the tumor which, once started, very quickly increases in size.

The first consequence of this emigration is the formation of a kink in the first part of the rectum at or near its union with the second part; making it more difficult, if not impossible, for the passage of the accumulated fæcal matter. This kink may be helped in its formation by a congenital abnormal state of the mesosigmoid and mesorectum.

Gas is the best dilator and it is rare that, even when the tumor has reached enormous size, there is complete obstruction. The gases prevent this, passing between the fæcaloma and the intestinal wall which they gradually stretch, dilating the lumen of the bowel to many times its natural size.

Both Dr. Metreaux, of this city, and I have noted "chimneys" in these tumors which admit the passage of gases and liquid fæces. The intestinal walls, often stimulated by drugs, do their best to shove on this mass; and without doubt the normal peristaltic movements are increased in these cases, at least at first. The results are multiple: a hard tumor forms, the walls lose their form, size, and thickness, their muscular coat is converted into a fibrous coat, sometimes nearly a centimeter thick, and the whole mass with its intestinal covering migrates to the right iliac fossa. The rectosigmoid segment is often 20 centimeters in diameter, and this for a distance of 25 to 40 centimeters, from the peritoneal fold (Perthes') of the first part of the rectum to the descending colon, rarely much farther, while the transverse colon may be dilated and full of liquid fæces, but without hypertrophy or degeneration of its walls.

When the transverse and upper part of the descending colon is dilated, with hypertrophy and degeneration of its walls, I would consider it a case of Hirschsprung's disease.

I hope I have made clear my idea as to the formation of a fæcal tumor in a sigmoid and rectum which was *not* defective at birth.

The cause. At first a little carelessness regarding the bowels with possibly a slight defect in Perthes' fold.

The effect, a megasigmoid forever. Because these mesos will never regain their length and tone, this intestine will never return to its normal size and the walls will never lose their thickness or regain their original elasticity. Under these circumstances new fæcal tumors form, time and time again, each time with more ease, in spite of care on the part of the patient. Please note that I am not writing about "megacolon" "congenital idiopathic dilation of the colon" or "Hirschsprung's disease," a malady of infancy and without doubt very distinct from megasigmoid. Distinct, because 80 per cent of our cases are in persons over 40 years of age, who declare they did not suffer in their childhood from intestinal stasis. I know cases that have suffered from the formation of fæcaloma for 20 years, but not one who had it from childhood.

An inflammation of the small intestine is called "enteritis," of the colon, "colitis,"

of the sigmoid "sigmoididitis," and of the rectum, "proctitis" and no one would ever think of calling them all one disease.

We have reached the era in which we study segment by segment and not the whole. This is the reason why I insist upon calling the disease in question "megasigmoid" or "megarectosigmoid" and *not* "megacolon," as I consider that another disease. I consider "megacolon" as different from "megasigmoid" as laryngitis is from bronchitis.

Pary in 1825, Billar in 1820, Barth in 1870, Peacock in 1871, Chapman in 1878, Gee in 1884, Bristame in 1886, Gaume in 1886, Futterer and Middelfort in 1886, Hughes in 1887, Hirschsprung in 1888–96, Walker and Griffiths in 1899, Consetti, in 1899, Fenwick in 1900, Duval in 1903, Arraga and Zubizarreta in 1905, Castre and Viñas in 1906, Bing in 1907, Bhane and Chaulet in 1908, Finney in 1908, Gaujaux, Chasagnar, Ginard Arauz, Alfaro, Centeno, Arana, Guinard and Cular in 1909–10, Grégoire and Duval in 1913, Palma and Viñas in 1915, and more recently Manuel Galdóz in his thesis of 1916, "Megacolon in the Adult," have all caused more or less confusion by trying to describe two distinct diseases under one name. I have not read the original papers but deduce this from different sources and especially from the Galdóz thesis.

The majority speak of megacolon as a disease of children and not of adults. The ages in Hirschsprung's 10 cases were one of 2, one of 2½, one of 3, and one of 11 months. The rest were between 7 and 10 years (Galdóz thesis).

Duval gives us the following statistics: "four cases properly called congenital, among them Hamann's case (7 months' fœtus); three in which the distention of the abdomen existed from birth; five which, although there was no distention, evacuated their meconium only after 4 or 5 days; thirty-nine cases in which the first symptoms were noticed in the first period of life; four only in adults." Dr. Galdóz, on page 47 of his thesis, goes on to say: "As one can deduce from the foregoing statistics, we are in frank opposition, as far as age is concerned, with the first symptoms of the disease, as in our nine cases, noted in the last 3 years, *all* were in adults; and if we add some more cases known to us, observed in other hospitals of this capital, I think we would come to the conclusion that this disease is evolutionizing, or that in our country, at least, the disease is most frequently found in adults."

I do not believe that there is any "frank opposition" at all. What I think is that Dr. Galdóz very cleverly describes megasigmoid with and without fæcal tumor and that not one of his cases corresponds in any way to Hirschsprung's disease. If there is no name for the disease described by Dr. Galdóz, then it is time to give it one.

If we study one by one the theories advanced by different authorities as to the pathogenesis of megacolon, we will be convinced at once that different authors have described different diseases under one and the same name. In no other manner can I conceive the inconsistency of their deductions.

Some say it is found only in fœtus (Hamann) and children during the first 3 years of life (Hirschsprung), while Duval gives 47 cases at birth or in first months of life, with only 4 of his 52 cases in adults.

In the records of the hospitals of this city during my work here (20 years), there are histories of over 300 cases, all in adults, and our city has a population of only about 250,000. This does not occur here only, for Galdóz reports the same findings as regards Buenos Aires as do also Drs. Viñas and Palma in their communications to the surgical society in that city.

There is another fact of importance and that is that in my cases I have seen only 1 per cent in which the fæcaloma formed outside the rectosigmoid segment (2 cases in 200), but Finney,[1] in 1908, summing up his paper on megacolon, says that up to that time no case had been reported as located in the rectum, and only 30 per cent were in the sigmoid.

Probably the truth is that megasigmoid with fæcaloma as *we* see it, does not exist in other countries, or if it does exist it occurs only rarely, while here with our relatively small population, congenital megacolon is very rare and almost unknown. The microscopical changes in the intestine may be the

[1] Surg., Gynec. & Obst., 1908, vi, 624.

same in both cases, but I do not think this is sufficient reason to consider them as identical diseases. If these patients had had a megasigmoid since birth, not one in a hundred would have reached the age of 45, which is the most common age at which fæcal tumors are found.

For these reasons I beg rightly or wrongly to claim that there is not even a similarity between the two diseases. One is a disease of infancy, the other of adults of middle age; one occurs in the transverse and descending colon, the other in the sigmoid and rectum, only affecting the rest of the large intestine accidentally; one never involves the rectum, the other nearly always.

ETIOLOGY

Megasigmoid is, in all probability, caused at first by neglect on the part of the patient, but once dilatation of the sigmoid and superior rectum begins, there are other factors which help the disease and prevent its easy cure. It is not a congenital or neuropathic factor which produces more megasigmoids in the Argentine Provinces of the interior than in other places, nor is it the water, the difference in food or way of cooking, the grapes with their seeds, the climate, occupations, houses, ways of living or want of healthy exercise. There is no more colitis or enterocolitis here than in any other place. In these provinces there has been a notable absence of water closets. Up to 15 or 20 years ago, a water closet with seat as ordinarily used today was unknown, even in rich men's houses, schools, or public buildings. Outside the towns, the vineyard, behind the barn, the hen-house, or a clump of bushes were the places in which everybody was obliged to defæcate, while in town a hole in and on a level with the floor in a little room at the far corner of the back patio, was the universal water closet. This hole led to a cess pool.

In Europe and North America, both in town and country, there has always been the water closet on more or less modern lines. It is the same in Buenos Aires and the Argentine Provinces.

True it is that squatting down in this way was undoubtedly the original manner in which primitive man defæcated. Had primitive man fæcal tumor? Probably no; the fight for life, more physical exercise, less modesty, and different food relieved him from the bothers of megasigmoid. After extended correspondence with colleagues from all parts of the world, I have come to the conclusion that laziness and neglect, united with the lack of private and proper places to defæcate are the chief causes for the greater number of such cases in Mendoza.

Every physician and surgeon knows that one of the causes of chronic constipation is the failure to answer the call of nature at the moment. A person has a desire to defæcate and for some reasons does not go to the toilet at once; probably he will not go to stool that day. These reasons are multiplied when there is no water closet within miles. If a young girl has to go to the corral or behind a bush to answer a call of nature, she will not always be able to do so when she likes, but must go when circumstances allow her to do so. Thus is formed the habit of constipation; little by little it becomes worse until it is necessary to take medicine to help the intestine now tired, dilated, and beginning to form an elbow or kink at the site of Perthes' fold, in the first part of the rectum.

Squatting to defæcate must, I should say, increase these kinks when once they are started, the angles become more acute and naturally it is more difficult for fæcal matter to pass, while, especially if there is constipation, the intestine itself has to work harder and the muscular coat begins to thicken with the resultant hypertrophy and later fibrous degeneration.

If, united to these logical common sense causes, the patient begins to take improper drugs (drastic cathartics, etc.), he will get a dilated hypertrophied rectosigmoid more rapidly. If there has been a slight congenital defect, in any case, the pathological changes mentioned will come on still more quickly.

Once the megasigmoid is formed, it is only a single step to a fæcal tumor, even if the person is careful. One day the kink is more pronounced, there is too little moisture in the excrement, the little pill or powder has been

forgotten, or easier still, forgetfulness, laziness, or want of opportunity has prevented the patient from emptying the bowls for 1, 2, or more days, with the result that he now requires hydraulic power (enema) or manual labor (finger) to empty the bowel. We must also remember that in this stage, there is diminution of reflexes and that these persons have lost, in a great measure, the desire to defæcate. This is one of the most notable features of these cases. Enormous masses of fæcal matter, weighing as much as 8 or 10 kilograms do not produce a desire to defæcate. From any book or physiology, we learn that the presence of fæcal matter in the lower bowel produces the reflexes which awaken the desire for this act, but, as I have said before, in these cases all reflexes are abolished, due in all probability to so often having been left unanswered. Marfan says (Thesis, Galdóz, 52): "Nothing proves that the dilatation is a primitive modification, as it has never been seen in an unborn child; and he considers that megacolon is a modification following congenital constipation. a disease which is closely related to multiple and exaggerated inflexions of the sigmoid flexure." Going on he says: "Stasis of the fæces would be sufficient to explain the series of consecutive phenomena which we observe in this disease. The dilatation is probably due to distention by the gases of putrefaction and, as for the hypertrophy of the walls, this would be at least in part dependent on the chronic colitis analagous to what occurs in the bladder when there is an obstacle to the free passage of the urine."

I believe Marfan had seen and studied a few cases of megasigmoid and not of congenital megacolon. I agree in every way with his hypothesis, and if we find a slight congenital defect in Perthes' fold and much indolence on the part of the patient, we can completely fill the picture.

SEX

The division as to sex is about equal.

AGE

It occurs most commonly between the ages of 35 and 50, is never found in children (my youngest case was a girl of 20, my oldest a man of 85).

SOCIAL POSITION

It is found in all social spheres and without relation to occupation. We see more poor than rich cases. Fæcal tumors are more common in the poor because the rich exercise more care to prevent their formation.

If a well-to-do person has once had a fæcaloma, he will take special precautions and really try to prevent the formation of others; as a rule with success, but he never cures his megasigmoid. The poor and generally indolent, after repeated extractions of fæcal masses, pay, in the long run, for their ignorance and laziness, with their life. In both rich and poor, megasigmoid shortens life.

PROGNOSIS

Megasigmoid without fæcal tumor never improves without surgical intervention. It is not necessarily fatal at once, but without doubt, as I have said above, it shortens life. There supervenes stercoræmia, obstruction, occlusion, volvulvus of the sigmoid flexure, gangrene of the same, perforation — spontaneous or as a consequence of manipulations or drugs for the stasis — peritonitis from that rupture or from a perforation which may be microscopical. Let us consider these in their order.

Stercoræmia or copræmia. This with its sequelæ is the most common outcome of megasigmoid. The condition is always grave and often leads to a fatal result. The absorption from fæcal stasis is pernicious to the general health, bringing on a state of anæmia and cachexia easy to recognize. It is really blood poisoning. The patient has a pale, earthy-colored skin, he loses appetite and flesh, and becomes so emaciated that he looks like skin and bone. The temperature varies from 35° to 41°C., but is low most commonly; it will vary many degrees in a single day. Weakness becomes extreme, the function of all organs is greatly impaired, and death follows in spite of all treatment. While the fæcaloma exists in the rectosigmoid, as a rule, the stercoræmia is not so grave, but the moment it is touched, moved or broken up,

extracted by finger, spatula, or other similar instrument, sterile water, medicated water or oils, the patient grows worse, fever comes on, and very often death results. In other cases and fortunately the most common, after the extraction of the fæcaloma, for a few days the patient has low fever in the afternoon and 35°C. in the morning. He takes castor oil and a daily intestinal wash by enema. With good food and care he is able to go to his home in 8 or 10 days only to return in 6 or 8 months, in spite of all care, with a new tumor. In indolent and ignorant persons the return will be in 4 or 5 months. Some come to the hospital with the fæcaloma and stercoræmia well developed. When this occurs, in spite of extraction, iliac anus, appendicostomy, cæcostomy, etc., death is the general termination.

Obstruction. Total obstruction or occlusion of the intestine is very rare. Gases always pass and generally liquid fæces. In fact, diarrhœa is often one of the first symptoms noticed by the patient when he does not even suspect a fæcaloma. The gases dilate the lumen of the bowel as the fæcal bolus forms, and this is always smaller in diameter than the intestine, for this reason. The occlusion is always due to kinks and without surgical aid the case terminates rapidly. A colleague claims to have relieved a case of occlusion by copious enema.

Volvulus of the sigmoid. I have operated on 9 cases of volvulus of the sigmoid. In two gangrene had already set in. If an operation is not performed in a few hours, death usually follows rapidly. In the cases I have seen, the megasigmoid takes a turn on itself, the meso acting as the center of the circle. In 7 cases there was a half and in the other two a complete turn of the gut. In one of each, gangrene was already present. In all the cases the turn was to the right, the enormous sigmoid passing in front of the rectum. The external border of the sigmoid instead of being in contact with the left wall of the abdomen and occupying the left iliac fossa, was in the right iliac fossa and in contact with the right lateral wall of the cavity. The moment volvulus takes place, there is acute and complete occlusion with the classical symptoms. There is also an enormous tumor in the right iliac and lumbar regions, which corresponds to the greatly dilated (with gas) intestine. Of my 9 cases only 3 were saved. I have seen at least 10 other cases of volvulus, but owing to their being admitted to the hospital in an utterly inoperable condition, no intervention was attempted and seldom was an autopsy done.

Gangrene of the megasigmoid is often a direct consequence of the above and rarely is found after manual extraction of the fæcal tumor. I have seen only one case from this latter cause; without operation it is, of course, fatal.

Peritonitis from perforation is fatal, with or without operation. Theoretically, operating at once ought to save some cases, but when this perforation occurs, the general condition of the patient prohibits intervention. The perforation is caused by gangrene in spots due to the incorrect use of instruments in the extraction of a fæcaloma or possibly by the use of drugs, although I have never been able to prove this latter by autopsy.

Peritonitis without perforation. There is a fatal peritonitis which comes on without perforation after the extraction of fæcal masses. It is caused, I believe, by absorption or filtration of poisons through the walls of the descending and transverse colon which are dilated and very thin and is *not* caused by absorption from the diseased walls of the megasigmoid.

DIAGNOSIS

Usually there is no difficulty in making a diagnosis, as the patient tells us what his trouble is. Occasionally, however, when there is diarrhœa, he does not suspect an accumulation of fæcal matter. The practical man should remember this and never fail to make rectal examination. He will feel a hard tumor in the left iliac fossa or in the middle line, varying in size according to the length of time it has been forming. The consistency of the tumor is pathognomonic; in no other tumors, is it possible to produce palpable indentations in the substance with the examining finger.

Another pathognomonic symptom found in fæcaloma with megasigmoid was first pointed

out to me by Dr. Alfred Metreaux who has also had a vast experience with this disease. If, in the presence of gases around the tumor, between the tumor and the wall of the intestine, the examining hand exerts pressure, the gas adheres to the tumor; on slightly relaxing the pressure one can feel the return of the gas with abrupt separation from the wall—often producing a perceptible sound.

Perhaps this is Gersuny's sign mentioned in Keen, *Surgery*, page 655, vol. iv, chapter by Van Hook and Kanavel.

Without the use of the X-ray, the proctoscope, or the sigmoidoscope it is not so easy to diagnose megasigmoid without fæcaloma. To give X-ray specialists an idea, in one case, afterward cured by operation, 4 liters of gum acacia water with 400 grams of carbonate of bismuth were injected into the dilated rectum, sigmoid, and colon without any difficulty and without the patient having the slightest desire to expel it.

I have never had an opportunity of using a modern proctoscope or sigmoidoscope, as used by specialists in rectal diseases. My opinion is that as the greater number of cases, when first seen, have a fæcal tumor and a decided kink or angle at Perthes' fold, the use of such a tube would be dangerous. And again, seeing a fæcal mass would not help either the diagnosis or the treatment.

Differential diagnosis. There are few diseases which a practical surgeon would be able to mistake for megasigmoid with tumor formed or megarectosigmoid: peritoneal bands, volvulus, kinks, intussusception, invagination, megacolon or Hirschsprung's disease, a foreign body in the rectal pouch, uterine tumor, pregnancy, ectopic gestation on left side with bleeding into the peritoneal cavity, or tuberculous peritonitis with liquid. All may, in certain cases, produce obstruction to the passage of fæces and gas; in all of them there would be more or less distention of the abdomen, but it should never be difficult to make a differential diagnosis. In any exceptional cases, if palpation will not clear up the diagnosis, the X-ray would be sure to do so.

Pregnancy with fæcaloma. I have had 5 cases myself and know of 5 more from the maternity hospital and from colleagues. Pregnancy with megasigmoid is a very serious condition. I believe the best thing to do in such cases is to produce an abortion or premature labor the moment the diagnosis is made, and then treat the fæcal bolus and its cause. In the maternity hospital, they have had three deaths from this cause in the last 3 years. The number of mild cases here with accumulation of fæces which yield to soap suds and castor oil treatment is larger proportionally than of those in the maternity hospital of Buenos Aires.

TREATMENT

Medical treatment alone is not only useless but it may cause great harm. In more or less than 60 per cent of my cases, the treatment found in the textbooks in the chapter on "coprostasis" is followed (Keen, vol. iv, p. 656): enemata using different solutions, a gloved finger, dilatation of the rectum at Perthes' fold, and straightening of kink just above, will generally bring the examining index finger in contact with the fæcal mass, which is broken up and hooked out with the same finger, while the left hand is pressed on the abdomen to keep the bolus within reach.

Sometimes the fæcaloma is so hard that the index finger cannot penetrate it. Then the handle of a large tablespoon, spatula, or best of all, one of the blades of a vesical calculus forceps will aid; the operator should always guide the instrument with the finger. Never, never use an instrument without guiding it with the finger and resist the temptation or advice to use any instrument *blindly*. Always remember that the second part and possibly the lower end of the first part of the rectum is thinner and weaker than normal, and that kinks are very easily wounded causing irreparable damage. I consider it much less dangerous to open the abdomen and attack the bolus *in situ* than to use any one of the instruments recommended for use beyond the reach of the guiding finger. Enemata, soap suds and olive oil, are used; another method is trituration, and so on.

Sometimes the patient or surgeon gets tired out. Then the operation can be held over and continued the next day, leaving a

good dose of oil in the bowel. It is surprising what can be done in this way. An injection of morphia just before starting or even rachis anæsthesia is necessary in some patients. I always use novocaine in first or second lumbar space. The anæsthesia makes it easier to push down the fæcal masses within reach. The next day castor oil and daily intestinal washing with sterile water and common salt are given (7:1000). After a week in bed I give medical directions for preventing the formation of another fæcal bolus, and discharge the patient. These are the benign cases, about 60 per cent, where the fæcaloma has formed in the last part of the sigmoid and first part of the rectum, where the mass is not very hard nor the disease of long standing.

In the cases in which the tumor is in the sigmoid and cannot be reached with the finger, I give large doses of opium and belladona to relax the upper rectum. Three days of these drugs combined, with soap suds and oil, have on several occasions allowed me to reach the mass with a finger and conclude the case as cited above. I have been advised to use the whole hand, but have never used more than two fingers. I believe the use of the whole hand, be it as small as you like, is very dangerous and have seen at least one death as an immediate result.

In cases where the tumor cannot be reached with the finger, after the use of opiates and enemata (including 50:1000 sulphate of magnesia, drop by drop), I operate by the abdominal route. The most common operation is extra-intestinal (coprotricia), i.e., trituration of the fæcal masses *in situ* without opening the wall of the gut. In cases where the abdominal walls are flaccid and thin and the fæcal masses not very hard, it is possible, under a general anæsthesia, rachis or chloroform, to break up the tumor without opening the abdominal cavity. I have been able to do this in five cases, once without anæsthesia, with good success, but I cannot recommend it. I believe the open operation is less dangerous and more satisfactory in every way.

The open operation most commonly used by me is that known as Llobet's, first performed by Dr. Andres Llobet about the year 1890 in the Rawson hospital, Buenos Aires. I saw him do it in 1896. He used a mid-line incision, whereas for the last 18 years I have used a left mid-rectus incision through the skin, then turn the rectus muscle inward, and open the abdomen underneath. No muscle fibers are cut. This, I think, makes a better and safer opening without danger of hernia later.

The incision should be long enough easily to admit the surgeon's hand, at least 10 centimeters. I put compresses on each border of the wound—Moynihan's tetra clothes are very good—and introduce my hand in the abdominal cavity. Beginning at the lowest pole of the fæcal bolus, I break off pieces and push them farther along the gut where they are thoroughly triturated until they are of the consistency of "boiled rice with milk." As this stage is reached, the mass is pushed on toward the lower rectum. Another piece is now broken off and the same process carried out until the whole hard mass has been well triturated and pushed on. The abdomen is then closed, with special care to close the rectus muscle over the inner wound.

Should the patient have been vomiting I inject into the bowel somewhere above the trouble, 30 grams of sulphate of magnesia in saturated solution, using a fine needle and putting a fine catgut stitch in the serosa for more surety.

A dose of castor oil is given the following morning, and the treatment continued as in the simple cases.

I have done Llobet's operation 15 times with 2 deaths, one a week and the other a month after the operation, both from stercoræmia. I have seen more fatal cases in other hands, but statistics in this class of cases are confusing and misleading. If the bad cases which have been sick are operated on, after trying, perhaps for weeks, to get the fæces out by natural outlet, more deaths will result than if only good cases are operated on. Again, if an attempt is made to break up very hard masses through an enormously dilated and diseased intestinal wall, and trauma and hæmorrhages are produced with perhaps gangrenous patches following, the patient is very likely to die. Perhaps the less said about statistics the better. Moynihan

said they could be made to tell anything "even the truth," which is better than another great man who said "they are lies—d——lies." I have done an autopsy on one of these cases and found considerable extravasation of blood in the intestinal wall, undoubtedly produced by too vigorous handling.

During operation I have often noticed slight hæmorrhage into the bowel wall which formed small hæmatomata but which have never, to my knowledge, interfered with a good result.

To conclude, Llobet's operation of coprotricia is very good, safe, and convenient, when done without undue vigor in handling the intestine and in cases which are at all operable.

Three times I have opened the abdomen as described, with the intention of doing a Llobet and have found a mass so big and hard and with a chronic megasigmoid and consequently a diseased fibrous wall, that I decided to extract the tumor *en bloc* by opening the intestine. I was afraid that gangrene would follow or serious hæmorrhage into the intestinal wall produced by the forces necessary to break up such a hard bolus. In all these cases the fæcaloma was situated in the sigmoid and encroached on the first part of the rectum. It was the shape of a football and all were about the same size, 50 centimeters in circumference. In these cases the meso is very long and in two I was able to operate outside the abdominal wall. The other was done within the wall. The following technique was used: first, the distended intestine was brought out of the abdominal cavity, tetra cloths were placed around it and the enormous tumor within. Second, an intestinal clamp with rubber-covered blades was attached above and below the fæcal mass to prevent the escape of fluids. If there is any difficulty in placing the clamp, a piece of gauze may be tied around with the same effect, taking care to make the smallest opening possible in the mesentery.

The bowel is opened in the line of its longitudinal band, making the incision with a single stroke and sufficiently long to allow the extraction of the tumor without having to touch it. Two hands are then placed under the mass which is easily delivered into the hands of a nurse. This maneuver is singularly easy; if the incision has been properly made, from 22 to 25 centimeters long in my cases, there is no trouble at all and no liquid escapes. The cavity is now wiped out with care and the suture begun. All the coats of the bowel are from 5 to 8 millimeters thick. The mucous coat is easily separated and seems bigger than the fibromuscular coat in which it has the appearance of hanging. Although thickened this coat was friable as in a normal mucosa. The serosa was not changed as the others but was very adherent to the muscular coat, which made the last line of sutures more difficult. Note that the greatest pathological change takes place in the muscular coat, making it thicker, tougher, and de cidedly more fibrous.

I used the following method in suturing this long intestinal wound: first, suture mucosa with No. 1 catgut using a Dupuytren continuous stitch with a cross every now and then; second, suture muscular coat with No. 1 silk, Cushing's continuous stitch "Greek border" with the stitches close together and with great care to get perfect apposition; third, make simple continuous catgut suture of serosa over silk suture, letting the needle go into the muscular coat; fourth, place another continuous catgut Dupuytren suture of the serosa inverting sufficiently to hide the last line. The suture of such diseased bowels must always be done with especial care to prevent accidents when the patient has the next bolus which, unless something else is done later on, is a certainty.

The fæcalomata in my cases weighed 560, 640, and 700 grams. All cases were operative successes. Two left the hospital fairly well, while one died a month later as a result of a second operation of which I will speak later.

Formerly, every 2 or 3 months, there would be admitted to the hospital a case of fæcal bolus with accumulation of fæces, occupying all the large intestine. Stercoræmia was well marked. In some I have done a right colostomy and twice I did an appendicostomy, but sooner or later all these cases died. I always use local anæsthesia, but in the presence of stercoræmia any operation gives 100 per cent

mortality. What happens is that once you move or try to move the masses of fæces with any liquid the absorption into the general circulation is greatly increased, and death comes more rapidly. They are really poisoned by their own excrement and cannot withstand even manual extraction. For some years I have given up trying to relieve hopeless cases by surgical means.

Let us consider the cases from which we have removed the fæcal masses and saved the patient for the moment from death by self-intoxication. Have we cured him? No! What is to be done? This is the problem. About 1913, I studied seriously all the literature obtainable and could find nothing to meet all the conditions present. In the hospital we tried all the short circuit operations and also total colectomy, following the technique described in the textbooks for them. The Lardenois technique for total resection of the colon is perfect to my mind, as is also that of Sir Arbuthnot Lane, but neither will answer in these cases. The diseased intestine extends as far at least as Perthes' fold, and it is impossible to make an anastomosis which will heal in diseased tissue. The patients themselves have no resistance so far as operations which require a general anæsthetic are concerned. They are not like megacolon (Hirschsprung) or chronic intestinal stasis (Lane); surgically, they resemble cancer of the upper rectum more than anything else generally written about. I believe that, in many cases, the best operation is resection of all the diseased part and either an inguinal or lumbar anus, or else the descending colon should be brought down and passed through the second part of the rectum to the sphincter ani. But there is a great difference between cancer and megarectosigmoid. In cancer the patient is always well nourished, and in good health otherwise to withstand a major operation, or else the case is inoperable (metastasis); while in megarectosigmoid, the patient as a rule is a very bad subject for any operation. His fæces have poisoned him, his flesh heals badly, and his intestine not at all. You can stitch with care, use adjacent serosa or make flaps of it, use the surrounding connective tissue, put a rubber tube high up in the bowel at a great distance from the anus, the utmost care can be exercised throughout, —and the result is the same.

I have seen more than one case in which an anastomosis had been done, and the patient was considered cured and out of danger as long as 20 days after operation, who suddenly developed an acute peritonitis, and postmortem examination showed non-union causing leakage of intestinal contents.

After bitter experience with a dozen different operations for megasigmoid with continual formation of fæcal bolus, I finally developed a technique which I have used in a limited number of cases—one which I believe to be original. It is analagous to Moynihan's operation *gastroplication* (Keen, vol. iii, p. 946) where I confess I got the idea. It should be called "sigmorectal plication" or "enteroplication."

I have done only 5 cases as yet and there was but one death from an accident not due to the operation. The four successful cases are all in better general health, and by laxatives and enemata which before operation were ineffectual, they can now prevent the formation of new fæcaloma. I am quite convinced that no "plication" operation is perfect in results, but in patients with megarectosigmoid I believe it to be the best, and especially as the general health of the majority of the patients prohibits a more radical operation without an enormous percentage of deaths. Any surgeon interested will readily understand the technique I use by referring to figure on page 946 of Keen, vol. iii. A nurse passes a rubber tube, an inch in diameter and fairly soft, into the rectum after rachis anæsthesia with 15 centigrams of novocaine (segun arte). The abdomen is opened through a left mid-rectus incision and the rubber tube passed into the healthy colon; the surgeon guides the tube through the dilated rectosigmoid. This tube is used as a guide in establishing future lumen of intestine and its use makes the "plication" much easier. I use silk for the first lot of sutures and apply them exactly as in Moynihan's gastroplication operation, about 1 centimeter apart, letting the needle go well into the muscular coat but never into the mucosa. With the fine point

of a thermocautery I now touch between stitches, going fairly deep. In my later cases I have used the thermocautery before putting in the first row of silk sutures. The object of this is obvious. When all the stitches are in place the tying is finished before I put in the second line of continuous simple catgut suture which covers over the silk knots. Finally I make a continuous Dupuytren catgut suture of the serosa alone which covers in the second line of catgut. The tube is withdrawn and the abdomen closed.

In my five cases the line of suture varied from 10 centimeters in length to 32 centimeters. All included the greater part of the first portion of the rectum. The longitudinal band of the sigmoid serves as a guide to the center.

VOLVULUS OF THE SIGMOID

The surgical treatment of volvulus may be found in any good modern book on operative surgery. I would like to state that I believe few of the authors have seen these cases for themselves but just go on copying the works of others. For example, in the presence of gangrene we are told to cut off the intestine and bring the ends of the healthy colon out of the abdominal wall as is done in the Mikulicz Brunns operation for cancer of the rectum, later closing the new anus by gradually uniting the ends with a special clamp, and finally closing the abdominal wall. These authors forgot that this is quite impossible here as the gangrene extends to the twist which in all my cases was so low down as to make the healthy rectal end too short to reach outside the abdomen. The upper end can be easily detached and brought out, but it is practically impossible ever to do as advised. The best treatment is to bring out the colonic end and close the rectal end; a future operation for uniting might be done. If the patient is in very good condition, the colonic end might be brought down to the rectal pouch or even lower, and an artificial anus need not be made. If there is no gangrene the operation can be best carried out, after reducing the volvulus, as recommended by Braun and Senn, i.e., by fixing to the left abdominal wall and shortening the mesosigmoid by folding it on itself and stitching.

PERITONITIS

Whatever the cause, peritonitis is fatal in these cases and surgery will not only not relieve but in many cases hastens the fatal outcome. Bitter experience has taught me this.

RECURRENT DISLOCATION OF THE PATELLA

With Report of Sixteen Cases

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A NUMBER of factors play important rôles in recurrent dislocation of the patella. Trauma is unquestionably important and frequent in the initial displacement. It is less important in recurrences. There is an inward twisting at the knee together with a blow or pressure on the outer side of the leg, and associated with this is always a strong sudden contraction of the quadriceps muscle.

Recurrent outward displacement of the patella is a lesion peculiar to young girls during their growing period and tends to persist into adult life. Usually the first displacement occurs between the ages of 12 and 18 years and this initial displacement is more painful and disabling than subsequent ones. Injury and excessive knock-knee are the chief etiological factors. The displacement occurs with the strong contraction of the quadriceps muscle, usually early in the act of knee flexion, since in ten to twenty degrees' flexion, the femur easily rotates inward on the tibia and an exaggeration of the knock-knee takes place.

There is a long line of contributing factors which aid these recurrences. Among these are found so many common postural conditions affecting the knee joint that it seems improbable that they alone can be the exciting etiological factor. The most constant contributing factors are:

1. Heredity. Abnormal mobility and laxity of the knee, associated with poor general posture are occasionally hereditary peculiarities.

2. Weakness of the quadriceps extensor muscle with resultant laxity of structures about the knee joint.

3. Static defects. Knock-knee has been long associated with this lesion but the frequency of knock-knee and the infrequent number of slipping patellæ rather weaken this as a strong etiological factor. It is necessary that the outer ridge of femoral condyles be poorly developed. Probably all postural defects act as factors only secondarily through this associated developmental defect.

4. Flat feet give static strain on the inner side of the knee joint and may contribute that additional twist which aids displacement.

5. Elongated patella tendon with relaxed synovial membrane and capsule, both usually due to chronic synovitis. The distention of the joint causes stretching of all the retaining structures.

6. Imperfect development in size and shape of the patella in relation to the external condyle will allow the patella to slip more easily to the outer side.

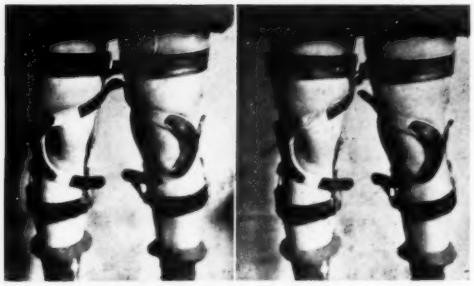
7. Imperfect development in the size and shape of the external condyle is important. Graser (9) did a supracondylar osteotomy in one case in which he felt this faulty relation required correction.

That the external condyle is occasionally underdeveloped and does not thereby offer a bony resistance to the displacement, is acknowledged by Albee (2) who has used a bone graft to correct this anatomic defect, tilting the outer rim of patellar surface of the femur to block the lateral thrust of the patella.

Of the above lesions, the lateral tendon attachment and the faulty condylar development are the most constant pathological findings. Static defects and capsule relaxation are important in releasing the normal factors restraining displacement.

DIAGNOSIS

The patient usually makes the diagnosis, giving the history of (a) severe initial displacement, (b) recurrences, (c) constant fear



Figs. 1 and 2. Jones knee brace with pad attached.

lest displacement recur—with resultant inactivity.

The initial attack is most painful. The knee is flexed and the patella is found on the outer side of the condyle. Following reduction, expecially in the initial attack, an acute synovitis develops.

TREATMENT

A. *Acute attack.* The treatment of the initial attack calls for immediate reduction of the displacement. This can usually be accomplished by sudden extension of the leg with pressure of one hand against the outer condyle, pushing the patella to the median line. The injury calls for immobilization by means of plaster for 3 weeks, and were this done following the original injury recurrences would be less frequent.

B. *Recurrent attacks.* The treatment of recurrent attacks may be divided into (1) supportive, (2) stimulative, (3) correction of static errors, (4) operative.

1. Supportive treatment in the earlier attacks usually gives the patient confidence. This may be obtained by the use of a split knee cap with a crescentic pressure pad to aid in holding the patella in place or it may require a Jones knee brace with a pad attached (Fig. 1). These should not be used without stimulative treatment, and if used too long are apt to cause varicose veins and œdema of lower leg from constricting straps.

2. Stimulative treatment consists of (a) baking and massage, which will to a degree give tone to the relaxed ligaments; (b) exercises to strengthen muscles and ligaments and to develop postural strength.

3. The correction of static errors is most important. In many of these cases there is marked abduction of the feet (weak or flat feet). This should be corrected in all cases by the Whitman type of foot brace. This correction counteracts in a mild degree a moderate knock-knee. In spite of conservative measures, the knee frequently remains not dependable. In such cases operation should be considered.

4. *Operative methods.* The operative methods are (a) capsulorrhaphy, (b) transplantation of part of patella tendon, (c) transplantation of bony insertion of patella tendon.

Capsulorrhaphy. Alone this operation has been a distinct failure. In conjunction with other procedures, it might be used, provided the length of time of the disability has been such as to cause the inner capsule to relax markedly and secondary deformities to develop. The authors have never done this, however, either alone or in conjunction with the following methods.

Transplantation of part of patella tendon. In the early cases of the authors, this procedure was followed, all cases showing good results. The objections, however, are—

1. Twisting of half of the patella tendon, which, although bringing more tautness between outer side of patella and external condyle, is open to the danger that relaxation of this tendon may take place. In general, a muscle cannot function in two places at once.

2. Splitting of tendon, sewing and handling ends cause them to fray and make it impossible to secure a clean, firm anatomic attachment, whether subperiosteal or into a bony cavity. The patella tendon is surprisingly thin and, on account of this tendency to fray, it is almost impossible to suture. We have, therefore, come to use only the bony transplant operations.

Technique of transplantation of patella tendon (Figures 3, 4, 5, 6 and 7). Usual two-day preparation. Tourniquet.

A curved incision is made from the inner side of the patella well down over the attachment of the patella tendon (Fig. 3). The skin is clamped off with towels. The patella tendon is exposed to view. An incision is made

Fig. 3. Drawing showing line of incision.

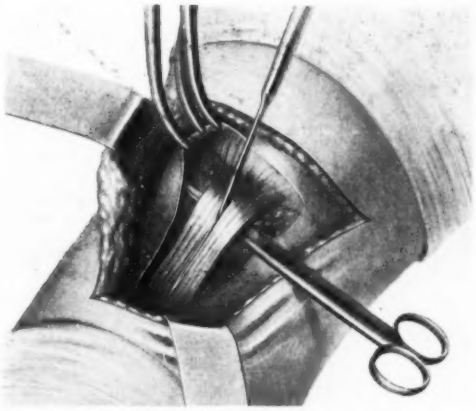


Fig. 4. Cutting outer half of patella tendon in slipping patella.

through the thin fibrous covering of the tendon which is then gently retracted and preserved as far as possible for resuture (Fig. 4). An incision through the center of the patella tendon is then made from the patella to the tubercle (Fig. 5), cutting it off at this point on the outer half. This half is then quilted with chromic catgut No. 3, turned in under the inner remaining half and sutured in a bony groove as far as possible to the inner side, on the inner face of the tibia (Fig. 6). The periosteum is then pulled over this insertion and the thin capsule pulled over these tendons (Fig. 7). The skin is closed with continuous catgut No. 2. Dry dressing is applied, followed by plaster from the toe to the groin. This plaster remains on for 10 weeks and is followed by gentle passive movements, baking and massage of thigh and calf, and increasing use. (See report of cases by authors, 1, 2, 3, 7, 8, and our cases 12, 14, and 15.)

Transplantation of the bony insertion of the patella tendon. (Figs. 3, 8, 9.) This procedure appeals from an anatomic standpoint as well as being practical and simple. The tendon is neither weakened nor traumatized. There is no suture through tendon and tendon strength is preserved. Results are excellent. (Cases from literature, 4, 8, our cases 13 and 16.)

Operative technique. Two-day preparation. Tourniquet.

A long curved incision is made halfway between the inner condyle and patella, curving over the tibial tubercle. The skin wounds are clamped off with towels, and the patella tendon with fascial covering is exposed. A light incision is made through the fascia from the inner border of patella to below the tibial tubercle; this covering is dissected, quickly clamped and retracted to right and left to be resutured later over the tendon. A wedge of bone the size of a thumb-nail is then removed, including the attachment of the inner half of the patella tendon. The patella tendon is split in half and a similarly symmetrical bony wedge removed from the inner surface of the tibia at a point that will take the tendon bone graft. The bony transplant is forcibly wedged into its new bed, the periosteum sutured with fine chromic gut, the light fascial covering resutured, and the skin closed with continuous catgut No. 2. Plaster is applied from ankle to groin and should remain on for 10 weeks. Following this, gentle motions, use, and massage should be conscientiously carried out.

CASE ABSTRACTS

Case 1. K. A., age 18, first seen March, 1915. The left knee cap had slipped out of place 3 or 4 years ago following injury to knee. Four months later while playing tennis "knee went out" again. Since then this has recurred at intervals. Physical examination was negative except for local condition. Any attempt to move left patella is received in a very

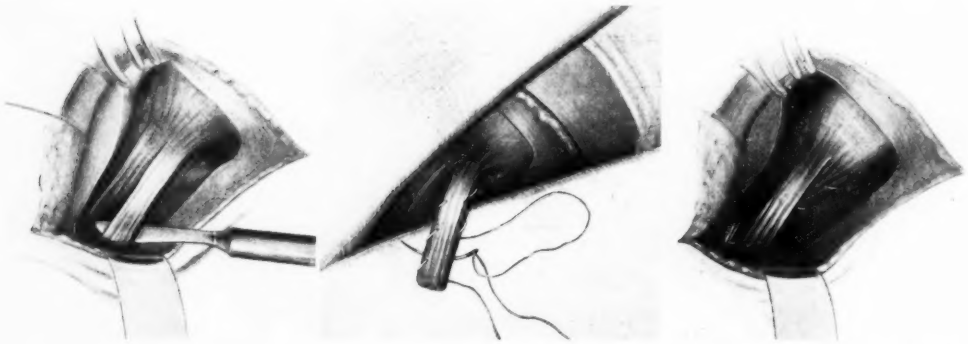


Fig. 5. Operation for transplantation of patella tendon. Note skin incision and the incision through the fascia covering tendon. Note longitudinal division of tendon.

Fig. 6. Running a line of silk sutures up and down the upper half of the patella tendon.

Fig. 7. Note the external half of the patella tendon has been sutured, passed under the internal half and is sutured subperiosteally. The wound is closed in the usual manner with continuous catgut suture.

apprehensive manner by the patient, especially when it approaches the external condyle. Slight pronation of both feet. X-rays of knee are negative.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of the outer half of the patella tendon into the periosteum of the tibia.

Result. Up to March, 1920 (5 years), there had been no recurrence. Perfect function.

CASE 2. Miss R., age 17, first seen August, 1912. Fourteen years ago patient fell against an oil stove and pushed the patella out. Since then this has slipped out at various intervals. Physical examination showed very relaxed capsule on the inner side. Any attempt to displace patella is very painful.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of the outer half of patella tendon into the periosteum of the tibia.

Result. Perfect function of knee. No recurrence to date (9 years).

CASE 3. T. K., age 23, first seen in April, 1918. About 5 years ago following an injury to the right knee, the right patella began dropping to one side. Has been treated with plaster casts, etc. Since the first attack the patella has slipped out seven different times. Physical examination was negative, except for local examination. The right knee is slightly swollen. All motions are limited and very painful. No surface heat.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of the bony insertion of patella tendon into tibia.

Result. Perfect function. No recurrence to date (3½ years).

CASE 4. I. H., first seen February, 1917. Since childhood has had a weak right knee which often twisted without warning, causing patient to fall to the ground, after which knee would be swollen and very painful. Following a fall, 3 years ago, right knee has been limited in motion. Physical examination was negative except for local condition. The right knee is swollen and tender. Patella approaches outer side of knee. Extension is normal, flexion is painful and limited. The patella slips to outer side of knee when knee is flexed. Some grating under patella on motion.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of bony insertion of patella tendon into inner side of tibia.

Result. Good function, no recurrence. January, 1921: "On the whole it seems to be greatly improved. There is no slipping of the knee-cap and, although it is somewhat weak and occasionally wrenches enough to throw me, it does not hurt as it used to and no swelling follows. I have a little more than half motion, but do not limp at all."

CASE 5. A. C., first seen June, 1911. Fifteen years ago patient fell through a piazza, striking on left knee. Since then has had attacks of patella slipping toward the outside of the knee, causing extreme pain and discomfort often lasting several hours. Last attack 2 years ago and since then knee-cap has been very loose. Physical examination shows left patella freely movable and can be carried well beyond median line on outer side.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Capsulorrhaphy (by another surgeon).

Result. (?) Case has been lost.

CASE 6. W. T., first seen July, 1906. Four years ago patient fell, striking on the outer side of the left foot, bending the knee inward. After that the knee has often "given out from under him." Feels insecure and has been wearing knee-cap and bandage.

The patella tendon is loose and attached toward the outer side of the median line of leg. It is also tilted down to the outer side of the knee. The

Figs. 8 and 9. Showing method of transplanting bony insertion of patella tendon.

internal condyle is prominent. The leg cannot be fully extended and there is slight crepitation under the patella.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of the outer half of the patella tendon into periosteum of tibia.

Result. Not satisfactory. Previous operation did not accomplish all that was desired so about 8 months later a rotation osteotomy was done just above adductor tubercle, the condyles being placed in their normal relationship. This operation resulted in normal function of the knee.

Case 7. B. D., first seen July, 1908. Knees have always turned in. Left knee has "given way under her" when walking. Has had several attacks during the last few years. Physical examination shows knees in semi-valgus position. There is also about 5 degrees' lateral mobility in full extension. Both patellæ are abnormally movable. The tibial tubercle is congenitally misplaced, being nearer the external edge of tibia than is normal.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of outer half of patella into periosteum of tibia.

Result. Immediate result good but patient has since disappeared.

Case 8. J. M., first seen November, 1904. About 12 years ago patient's knees began to "give way" from beneath her. The attacks came suddenly and she would fall. These occurred at intervals of weeks, always associated with swelling of the knee and "it always felt as if something came out from the inside of her knees." Physical examination was negative, except for local condition. Motions of knees are normal. On complete flexion, the patellæ move toward outer aspect of knee. Patient has a slight knock-knee.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of the outer half of patella tendon into periosteum of tibia.

Result. Normal function. No recurrence to date (17 years).

Case 9. Mrs. T. C. K., first seen March, 1912. Patient has had trouble with her feet for years. At the age of 10 years her left knee began to slip out at frequent intervals. Six years later her knee was operated upon by another surgeon, a reef being taken in the capsule. The knee remained well for 4 years when it began to slip out again in spite of the leather braces and knee-caps she had been wearing.

Diagnosis. Slipping patella.

Treatment. Capsulorrhaphy (by another surgeon).

Result. Failure.

Case 10. A. B., first seen August, 1915. About 19 years ago, left knee-cap slipped out for the first time. This has since recurred at frequent intervals. Four years ago, this knee was operated upon for this condition. Several hours (?) ago the patient tripped, fully flexing the knee. The patella was displaced laterally forcing the femoral condyles through the inner capsule, the tear being from the inner tuberosity and the front of the tibia. Patella tendon is intact.

Diagnosis. Recurrent dislocation of left patella and ruptured internal lateral ligament.

Treatment. Light plaster cast. Operation later.

Result. Good function.

SUMMARY OF AUTHOR'S CASES

No.	Name	Date First Seen	Original Injury	Static Defects	Treatment	Result	Remarks
1	K. R.	Mar., 1915	Slight trauma	Weak feet.	Tendon transplantation	Good	No recurrence
2	Miss R.	Aug., 1912	Dislocation of patella	?	Tendon transplantation	Good	No recurrence
3	T. K.	Apr., 1918	Contusion	?	Tendon bone transplantation	Good	No recurrence
4	I. H.	Feb., 1917	Twist	?	Tendon bone transplantation	Good	No recurrence
5	A. C.	June, 1911	Contusion	?	Capsulorrhaphy	?	
6	W. T.	July, 1906	Contusion	?	Tendon transplantation	Poor	Eight months later osteotomy done and condyles placed in normal relationship. Good result then.
7	B. D.	July, 1908	None	Tibial tubercle misplaced outward.	Tendon transplantation	Patient lost track of	Immediate result good.
8	J. M.	Nov., 1904	None	Slight knock-knee.	Tendon transplantation	Good	No recurrence
9	Mrs. T.C.K.	Mar., 1912	None		Conservative capsulorrhaphy	Poor	This case previously operated upon by another surgeon and capsule reefed without improvement.
10	A. B.	Aug., 1915	None	None	Conservative	Good function	
11	Mrs. T. S. M.	Oct., 1919	None	Flat feet	Conservative	No improvement	Operation later
12	E. H. T.	Oct., 1904	Contusion		Tendon transplantation	Good function	No recurrence
13	L. D.	Nov., 1919	Twist	None	Bone transplantation	Excellent	No recurrence
14	L. A. S.	Oct., 1911	Contusion		Tendon transplantation	Function normal	No recurrence
15	M. H.	Nov., 1908	None	None	Tendon transplantation	Function normal	No recurrence
16	Miss M. R.	Aug., 1920	Contusion	None	Bone transplantation	Function normal	No recurrence

CASE 11. Mrs. T. S. M., first seen October, 1919. For the last 24 years both knee-caps have been slipping out. These attacks come on very suddenly and are very painful. The patient has been wearing supports for the knees. Physical examination shows both feet slightly flattened with a moderate hallux valgus. The patient is wearing very tight split knee-caps which are causing swelling of the lower legs.

Diagnosis. Slipping patellæ (double).

Treatment. Conservative. Operation later.

Result. No improvement. Refuses operation and still has recurrent displacements.

CASE 12. E. H. T., first seen October, 1904. About 4 years ago, patient fell on knee and was confined to bed for a month with leg in splints. Two and one-half years ago she fell again, and remained in bed 2 months. Three months ago, she again fell and had to push patella back in place. Cast worn one month. Physical examination negative except for local condition. The entire musculature of the right leg is flabby and the power in this limb is considerably diminished. Both patellæ are more movable than normal.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of outer half of the patella tendon into periosteum of tibia.

Result. Function normal. No recurrence to date.

CASE 13. L. D., first seen November, 1919. Five years ago patient twisted the left knee. Laid up a week. One year later fell again, this time striking on the right knee. A year later her knee slipped out five or six times, always accompanied by pain and swelling. Physical examination shows a moderate amount of fluid in the right knee and any attempt to push the patella to the outer side is very painful.

Diagnosis. Slipping patellæ, and operation is recommended.

Operation. Transplantation of the bony insertion of the patella tendon in tibia.

Result. No recurrence; excellent function; no subjective symptoms.

CASE 14. L. A. S., first seen October, 1911. Nine years ago the patient injured the knee which became swollen and painful. Patient was unable to walk. Knee-cap has slipped out twice in the last 2 years. Physical examination shows patella unusually mobile. Can almost be thrown off to outer side with ease.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of outer half of patella tendon into periosteum of tibia.

Result. No recurrence. Function normal to date.

CASE 15. M. H., first seen November, 1908. Left knee-cap has slipped out many times within the last 3 years. Physical examination was negative except for local condition. Left knee is moderately swollen. Fossæ on either side obliterated. Slight increase in surface temperature. Complete extension of knees possible with difficulty. Both patellæ tendons are elongated, those on the left more than the right. Tibial tubercle is external to mid-line of patella; the pull of the patella tendon is, therefore, obliquely outward, the patella being tilted.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of outer half of patella tendon into periosteum of tibia.

Result. No recurrence. Function normal.

CASE 16. Miss M. H., age 28, first seen August, 1920. In 1915, patient struck right knee against a chair sustaining an injury which kept her in bed for

6 weeks. After that she was unable to use the knee even with a knee-cap and "it kept slipping out." Following each attack she was "laid up" from 6 to 8 weeks. Last attack July 7, 1920, following which she was confined to bed until September 6, 1920. Physical examination was negative, except for condition of right knee, which is swollen. All motions are painful, especially on moving the patella outward.

Diagnosis. Slipping patella, and operation is recommended.

Operation. Transplantation of bony insertion of patella tendon.

Result. Excellent function.

BIBLIOGRAPHY ABSTRACTS

Goldthwait in February, 1904, reported 11 cases of slipping patellæ with the following results: 1 case where tendon was transplanted with its bony insertion with good result; 5 cases where tendon was transplanted into periosteum with good result; 2 cases where tendon was transplanted into periosteum, not good result; 2 cases where capsule was quilted with not good result.

Krogius in *Zentralbl. f. Chir.*, Mar. 5, 1904, reported 2 cases (one double) where patella was drawn outward by tense outer portion of the capsule. He operates as follows: (1) approach the knee by Kocher incision; (2) incision from slightly above the patella, down a few inches in front of its outer edge to insertion of ligamentum patellæ; through the iliotibial band, tendinous expansion of vastus externus and fibrous capsular wall; (3) formation of bridge-shaped flap on inner side of patella connecting below with the tendinous expansion of vastus internus and fibrous capsule and above with muscle and fascia; (4) transplantation of the flap, left attached at both sides, across patella at its outer edge.

Result. First case: after 6 months patella slipped out again. Second case: result perfect after 3 months.

Whitlock in *British Journal of Surgery*, July, 1914, gives technique as follows: (1) reefing medial side of capsule with or without opening joint; (2) transplanting insertion of patellar ligament medially; (3) reinforcing the patellar ligament by grafting tendon of gracilis into it.

Dumferline in SURGERY, GYNECOLOGY AND OBSTETRICS, April, 1912, describes his technique as follows: patellar ligament is split, portion is turned up and sutured to the cut end of the tendon of the semitendinosus with chromic catgut. Capsule and fascia are then reefed.

Murphy's technique is given in Murphy's *Clinics* vol. iii, No. 4, August, 1914, as follows; Joint exposed freely by two longitudinal incisions, one on either side of the patella, turning the patella with the ligamentum patellæ to one side. Segment of bone between condyles removed to deepen the groove. Flap of fat and fascia then turned in from above and sutured over the denuded bone area to prevent ankylosis of patella to femur. Patella then replaced.

Graser in *Deutsche Gesellsch. f. Chir., and Zentralbl. f. Chir.*, July 9, 1904, reports a case where outer condyle of femur stood considerably farther backward than inner condyle when leg rotated outward. He operated by doing a supracondyloid osteotomy of femur and twisting the condyles so as to bring outer portion forward and inner condyle farther back.

Albee in *Orthopedic and Reconstruction Surgery* describes his technique as follows:

"A semilunar skin incision is made at the outer border of the patella sufficiently long to reach below the tibial tubercle and to a point above the external condyle. Avoiding undue disturbance of the underlying joint structures, the external condyle is penetrated with a broad thin osteotome on its external surface, making a bone incision from 1½ to 2 inches long, and situated about 1½ to ¾ inches below its anterior articulated surface and nearly in line with the long axis of the femur. This bone incision allows the anterior surface of the external condyle to be elevated to a plane above the internal condyle, by producing a greenstick fracture near the intercondylar groove, the object being to place a permanent and rigid obstacle in the way of outward displacement of the patella.

"When the anterior segment of the external condyle has been obliquely elevated to a sufficient degree to secure the desired obstructing effect, the width of the bone-gap thus formed is measured, and a section of bone sufficiently large to fill this cuneiform opening is removed from the crest of the tibia through the lower portion of the original skin incision, extended below the tubercle for this purpose. This bone-graft wedge can be easily and quickly procured by the use of the motor-saw. Before the graft is removed, it is drilled obliquely in one or two places with a motor-drill so that it may be pegged to the under portion of the external condyle after it has been put in position. Dowel pins, made from an additional portion of the bone removed from the crest of the tibia at the time the graft is obtained, and rounded by the motor-lathe to fit the drill holes in the graft, are driven into place.

"The cancellous structure of the condyle is easily penetrated by the bone-graft pins, but if any difficulty is encountered, the motor-drill can again be inserted into the holes already made in the graft and these prolonged into the external condyle. The ligaments and tendinous expansions are sutured over the graft with kangaroo tendon, thus securely holding the elevated portion of the condyle. The skin wound is closed with a continuous mattress suture of plain catgut, without drainage, and the leg from toes to groin is encased in a plaster-of-Paris splint in which it is allowed to remain for 3 weeks; at the end of this time passive motion and massage are begun.

"The advantages of this procedure are that with no sacrifice of joint cartilage, a minimal amount of joint injury is produced at the time of operation, thereby greatly lessening the dangers of limitation of motion and the formation of adhesions, and that the permanent blocking of any further tendency to displacement of the patella is effected by the actual elevation of the external condyle or an actual restoration of the normal mechanico-anatomical conditions. The soft parts are not interfered with. The only further suggestion in the case of extremely lax and stretched internal capsular ligaments is their plication with kangaroo tendon; this, however, is usually unnecessary, for if the external condyle is propped well forward, all requirements are fulfilled."

BIBLIOGRAPHY

1. TUBBY, A. G. Deformities Including Diseases of the Bones and Joints. Vol. i, chap. ii, p. 313.
2. ALBEE, F. G. Orthopedic and Reconstruction Surgery. Philadelphia: Saunders Co., 1919, p. 624–630.
3. GOLDTHWAITE, JOEL E. Slipping or recurrent dislocation of patella. Boston M. & S. J., 1904, cl, 169.
4. Idem. Permanent dislocation of the patella. Ann. Surg., 1899, Jan.
5. KROGIUS. Zentralbl. f. Chir., 1904, Mar. 5.
6. WHITLOCK. Brit. J. S., 1914, July.
7. DUMFERLINE. Surg., Gynec. & Obst., 1912, April.
8. MURPHY. Murphy's Clinics, 1914, iii.
9. GRASER. Deutsche Gesellsch. f. Chir. and Zentralbl. f. Chir., 1904, July 9.

AN ENDEAVOR TO EVALUATE CHRONIC SEPSIS IN PREGNANCY

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PART I

THE study of acute infectious disease has shown that certain types of bacteria which gain entrance to the body cause a sequence of clinical symptoms which may be said to describe the type of the invading organism. In most of these disease entities, the moment of invasion is a silent moment, followed by a period of incubation, ending in symptoms which go to make up the particular disease entity. There are many of these clinical entities. In some cases the invading organism is known, in others it is unknown. The definition of the clinical entity is not, however, dependent on the isolation of a definite bacterium.

In the following paper I wish to describe a clinical entity, apparent in pregnancy, which has chronic sepsis as its etiological basis. This clinical entity instead of describing a definite bacterium shows plainly that it is produced by many different kinds of bacteria. It is the singleness of the principle involved which demonstrates the entity. The manifestations like the bacteria involved are multiple.

In the study of disease the importance of the point of entrance of bacteria and the moment of invasion has been emphasized by MacKenzie in his book on the *Future of Medicine* (1). With the knowledge we have of the damage done to the human system by the invasion of acute infections, it is difficult to understand why the importance of an area of chronic infection, however small, is so little appreciated. If we regard an area of chronic sepsis purely as a point of entrance of bacteria within the system, the sequences which may follow can not be less harmful than those from the point of entrance in an acute invasion, provided it is admitted that the bacteria contained in the area of chronic sepsis can migrate to other parts of the body. Then the analogy becomes complete. Acute tonsillitis never killed any one, but it is the endocarditis or what not, that is the sequence of the tonsillitis, which kills. Just so the chronically infected tonsil or the abscessed tooth root does not kill, but in its primary element as a point of entrance of bacteria it has the same potential characteristics. That bacteria contained in these areas of chronic sepsis do migrate is too well settled today to warrant further discussion.

It is my belief that obstetrics offers a better opportunity for the study of the effects of chronic sepsis than any other branch of medicine. This belief is based on the conception that the processes involved in pregnancy act as the most severe physiological function test that the human system has to endure. As such it tends to bring out the previously existing weak points in the individual. It uses up a part or the whole of the reserve power of the different organs involved and thereby shows the defective organ when the reserve power of that particular organ is exhausted. I believe that the additional load of the pregnancy actually lowers the resistance of the maternal organism, if it is attacked by infection and that the effects of chronic infection already in the system are made more evident as a result of this reduction of the total bodily resistance. The high mortality among pregnant women in the influenza epidemic is consistent with this conception. The aggravation of many previously existing pathological conditions is also consistent.

With this conception as a foundation, already existing infection in the pregnant woman becomes of importance. Not only is it important in the individual pregnancy but also in the patient's whole obstetrical history. In drawing a conception of the effects of chronic sepsis in pregnancy, it is necessary to conceive of a clinical entity which covers months and years, since the etiological factor is often present for a like period of time. A great diversity of manifestations is likewise to be expected. In both of these characteristics the analogy to syphilis, the best under-

stood of the chronic infectious disease entities, is apparent.

Syphilis shows us that the chronic infecting agent can be persistently present in the system without causing symptoms of sufficient seriousness to send the patient to the doctor, yet this same infecting agent can show damage in some form which is often apparent only in pregnancy. The baby born with spirochætæ in its liver received those spirochætæ from its mother's blood stream and it doesn't matter whether the mother had the symptoms of syphilis or not.

What are some of the more common complications which confront the obstetrician? Toxæmia of pregnancy in its many forms is by far the most common and most important. Of this I shall speak later. Breast abscesses, non-suppurative mastitis, pyelitis, phlebitis, with an occasional pulmonary embolus are ever present dangers which must be constantly kept in mind by the obstetrician. These complications all have the common etiological basis, infection.

When it was first discovered that the typical case of puerperal septicæmia was due to extraneous infection, that is, that the infection was introduced at the time of delivery, the opinion naturally arose that all other infectious complications were the result of infection introduced from outside. As a sequel it has been universally believed that the breast infection was the nurse's fault in not keeping the nipples clean, that pyelitis was due to an ascending infection from urethra to bladder and thence along the course of the ureter to the pelvis of the kidney, and that phlebitis was thought to be universally due to parametritis with direct extension to the femoral vein.

There is much evidence available which tends to qualify this former conception if such cases are viewed from a broader standpoint. If we consider breast abscess as an infection of hæmatogenous origin and regard this abscess merely as one manifestation of a larger entity, chronic sepsis, the truth of the situation is more apparent. If we cease to regard the breast abscess as a complete clinical entity and view it as a symptom, a manifestation of the presence of occasional septic emboli in the blood stream, it will soon be apparent that many such cases will show other evidences of septic emboli or the effects of chronic sepsis in the system.

For instance, a patient had several non-suppurative mastitis attacks in her first pregnancy and these were *repeated in her second pregnancy*. She gave a history of frequent tonsillitis and had several attacks while under my charge. Physical examination showed two large diseased tonsils and a mildly decompensated mitral stenosis. There was a mild toxæmia present at the end of her first pregnancy.

The chronic endocarditis speaks for blood-borne infection in the past, probably originating in the tonsils and the repeated breast attacks in two pregnancies (always associated with high fever and malaise) point to blood-borne infection at the present time. The toxæmia is also a part of the picture.

Phlebitis, which is essentially a periphlebitic process resulting in thrombosis, is seldom unattended by other manifestations of hæmatogenous infection, past or present.

Pyelitis has been an ever-increasing complication of pregnancy in my own experience, and it has been impressive to note the number of such cases which present the history of pustular eruptions or furunculosis both before and after the appearance of the pyelitis. In such cases the pyelitis is merely an incident, a boil in the pelvis of the kidney. It is granted that these several complications of pregnancy are also sequelæ of infection when introduced from outside, but it is my contention that this sequence is merely clinical proof of the principles involved in the larger entity of which a focus of chronic sepsis is the continuous etiological factor.

With the conception clear in mind that a focus of chronic sepsis does throw off bacteria into the blood stream, not as a bacteriæmia or septicæmia, but as a series of bacterial emboli, it will be seen that the placenta, an organ constantly bathed by the maternal blood, is continually exposed to attack by these septic emboli, and therefore becomes an organ of great importance.

As a removable organ, it should present on its surface a record of damage done to it by

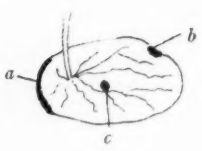

Fig. 1.

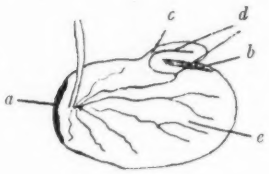

Fig. 2.

these septic emboli and these points of damage should have a chronological relationship to clinical evidences of acute infection elsewhere in the body. In a previous paper (2) I have submitted evidence that the infarct is infectious in origin. I also maintain that there is a chronological relationship between the time when the infarct is originated and the activity of sepsis, either acute or chronic elsewhere in the body. This point is so important that I desire to be more explicit.

The method of determining when an infarct originated, that is, the time in the pregnancy, can be determined in the case of infarcts which are on the edge of the placenta.

The normal placenta enlarges concentrically with the point of cord attachment in the center. During the growth of the placenta an injury to the edge (infection followed by localized thrombosis) stops the growth or enlargement of the placenta at the point of injury. Such a process results in an asymmetrical placenta. In the asymmetrical placenta the edge farthest from the base of the cord is the last placental tissue grown. In the normal placenta the whole circumference is the last tissue produced. In the asymmetrical placenta, by dividing the distance from the base of the cord to edge of placenta farthest away, the radius of a circle is found which would roughly represent the circumference of that placenta at 4½ months.

The position of the infarct within or without this circle will determine roughly the time in the pregnancy when it began, if it is on the edge, or, when it is in the middle, the time after which it must have occurred.

For instance, an infarct on the edge, Figure 1, *a*, at 3 months which stops the growth of the placenta at that point can not have occurred later. An infarct occurring at point Figure 1, *b*, could not injure the placenta at that point until the placenta had enlarged to that size which would from its position mean later than the middle of pregnancy. Likewise if an infarct occurs in the middle of the placenta at Figure 1, *c*, this injury may have occurred at any time subsequent to the growth of that part of the placenta which from its position in the diagram can be roughly estimated from 3 months on.

By observing the course of the blood vessels on the fœtal surface of the placenta, this method of growth of the placenta and the results of infarct formation on its edge can be demonstrated.

Figure 2, a diagram of the placenta in one of my cases, is an example. Figure 2, *a*, infarct formation which occurred within first 3 months and stopped further enlargement along the edge. Figure 2, *b*, infarct formation on edge of placenta shortly after the middle of pregnancy. Figure 2, *c*, a tongue of good placental tissue which grew out and around the edge damaged by infarct, Figure 2, *b*. Figure 2, *d*, course of blood vessel which originally pointed to region covered by *b* but changed its course to supply the tongue of placental tissue *c*, showing that placental tissue *c* grew subsequently to the infarct formation at *b*. Figure 2, *e*, enlargement of the placenta in asymmetrical formation to compensate for its inability to enlarge concentrically at region *a*.

This case demonstrates that infarct formation when on the edge of the placenta stops the growth of the placenta at the injured point. The position of these infarcts as noted was chronologically coincident with clinical symptoms in the patient's pregnancy.

Case 1. Primipara. Acute appendix removed August, 1918. Last period March 3, 1919. On April 1, 1919, there was slight bleeding which continued for several days. May 2, 1919, nausea, specks before eyes, and dizzy spells. June 1, 1919, another bleeding spell of one day duration. Dentist had been treating a devitalized tooth twice a week preparatory to filling nerve canals. Last treatment 3 days before bleeding began. X-ray showed an apical area. During the last part of August, 1919, she had an acute throat with white patches on tonsils.

Vomiting continued until motion was felt. Occipital headaches, specks before eyes, and œdema of legs until the the seventh month. October 28,

septic tooth extracted. November 10, no more specks or headaches. Sleeps better, feels better, œdema less. December 6, normal delivery of 7-pound baby. There was no rise of blood pressure or urinary disturbance throughout pregnancy.

The stirring up of the septic tooth chronologically preceded the bleeding in the early months of pregnancy and with the infarct formation at *a*, Figure 2. Infarct *b*, Figure 2, was formed also coincident with the attack of tonsillitis in August. No external bleeding, however, resulted. Toxic symptoms subsided after extraction of septic tooth. For other evidence of hæmatogenous infection in system, note appendicitis in August, 1918.

The following case had best be entered here on account of the similarity in the etiological factor.

CASE 2. A primipara had her last period on December 25, 1920. January 20, 1921, she had the sensation of approaching menstruation but did not flow. January 28, 1921, went to dentist who opened root canals of septic tooth preparatory to treating. X-ray film taken on that day showed wire in nerve canal and penetrating apical area. Also shows apical area on tooth adjacent. February 1, 1921, felt fullness in pelvis, had headaches and dizziness, and lost appetite. February 8, 1921, the untreated septic tooth was extracted. Symptoms during February: Three severe headaches associated with specks before eyes and blurred vision, nausea without vomiting. March 1, 1921, these symptoms stopped suddenly. March 16, 1921, first seen by me, at which time she felt perfectly well. Vaginal examination revealed a soft uterus, still in the pelvis in good position but not as large as the date would signify. March 23, 1921. Miscarried and was curetted at her home by another doctor who told her that he thought the pregnancy had been dead some time. Her tonsils had been removed in 1910.

Patient was a trained nurse and her observations reliable. The sequence of events in this case is most striking. The stirring up of the focus of sepsis undoubtedly threw bacteria into the blood stream which reached the placenta, injuring it sufficiently to terminate the pregnancy gradually. The cessation of symptoms suddenly about March 1 probably marks the time of the termination of the life of fœtus. I have seen a similar event in two cases of death *in utero* in a severe toxæmia at 7½ months. The miscarriage did not represent the time of the termination of the pregnancy but merely the casting off of the already dead fœtus.

CASE 3. A primipara came in for treatment of acute tonsillitis stating that she was 3 months pregnant. Two days later I was called on account of uterine bleeding which lasted five days.

CASE 4. A primipara while under my charge developed a tonsillar abscess which opened and drained spontaneously several times, followed in one week by uterine bleeding at the second month. Left femoral phlebitis also appeared on the tenth day after delivery with an afebrile convalescence up to that time.

These two cases are cited to show the sequence between infection in throat and uterine hæmorrhages. Both of these cases occurred previous to my association of these events with infarct formation and the placentas were not observed when they were delivered at full term.

CASE 5. A multipara developed an acute exacerbation of a chronic tonsil (scrapy throat, tenderness in glands at angles of jaw and malaise *without inflammation* of surface of tonsils) followed by miscarriage and curettage ten days later.

In this case the damage to the placenta by the infection was sufficient to terminate the pregnancy.

CASE 6. A primipara with large tonsils (right larger than left) and one devitalized tooth with history of abscess on it. She had uterine bleeding at 2½ months. Placenta was of the asymmetrical type with cord attached near edge and infarct formation along this near edge.

No clinical event associated in her memory. Patient not seen until eighth month.

CASE 7. A multipara flowed one day 6 weeks after her last period. No clinical event associated. Placenta was a typical asymmetrical battledore type. Patient had an enlarged heart with thrill and systolic murmur at apex. Tonsils diseased. Blood pressure rose to 140–100 in seventh month and she carried a pulse rate of 100–112 during last 2 months. Had a bleeding spell in second month of first pregnancy; also albumin in urine during the end of first pregnancy.

Although there was no clinical event associated with the bleeding in this case, her past history with the heart damage shows evidence of a previous blood-borne infection with the tonsils, the most probable etiological factor, still present. The bleeding spell was recorded by the shape of the placenta.

CASE 8. A multipara whose last period was July 4, 1920, had a very severe cold on election day, November 2, 1920. Her placenta showed a large

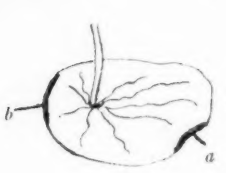

Fig. 3.

Fig. 4.

nfarct on the edge at about 4 months. No history of bleeding.

Previous history. First baby in 1918. Vomited throughout this pregnancy. Had a lot of trouble during this pregnancy with a painful carious molar. Molar was not removed until October, 1918, several months after the birth, when she had a miscarriage of twins on the next day. Bleeding had been present before the extraction, however.

Another miscarriage came in February, 1919. Her tonsils had been clipped 15 years ago and she still had tonsils in 1920 when I attended her.

This case raises the question which was the cause of the trouble. The severe cold was associated with the infarct in her last pregnancy without any septic teeth being present. The activity of the tooth in association with the vomiting in the first pregnancy and the first miscarriage tends to connect these events with the tooth. The second miscarriage might well have come from the clipped tonsils, which are notorious sources of chronic sepsis. The case demonstrates one important principle in the relation between treatment and results. The eradication of chronic sepsis must be complete to get results.

CASE 9. A primipara with large tonsils, the right larger than the left and with a history of stiff necks and stiff joints had a bleeding spell on February 8, 1921, 5½ months after her last period. In this case I had the satisfaction of making a note to look for the infarct at the time of birth, and it was there on the edge in the proper position in the placenta at *a*, Figure 3. No clinical event was associated with infarct *b*, Figure 3, which must have occurred at about 3 months.

CASE 10. A multipara with the following history. First baby July, 1917. Baby died in 5 days of hæmorrhagic disease. Weight 4 pounds, 11 ounces. Full term. March, 1919, miscarried at 3 months. March 5, 1920, complaint, cannot get pregnant. She had a mild leucorrhœa which responded to treatment. May 11, 1920, blood pressure found to be 130–100. She had one dead tooth with history of abscess one year after filling. X-ray showed abscess still present, also one other dead tooth. After removal of abscessed tooth, pimples on face ceased. Pregnancy took place in September, 1920. During latter half of October the remaining dead tooth was closed after treatment. December 2, 1920, had considerable pinkish mucus from vagina followed by a thick mucoid leucorrhœa. Blood pressure 118–80.

Note made to look for infarct at 3½ months. It was there resulting in a placenta shaped as diagram. Baby weighed 9 pounds, 4 ounces.

The small full-term baby which died of hæmorrhagic disease followed by a miscarriage was all subsequent to the history of the abscessed dead tooth. The cessation of the pimples on her face after the removal of this tooth suggests the removal of the source of the hæmatogenous infection. The infarct formation at *a*, Figure 4, can be attributed to the closing of the questionable dead tooth, as it is associated in time.

CASE 11. A primipara whose early pregnancy was not marred by nausea or vomiting and whose teeth were above reproach, had a severe attack of influenza about the end of her sixth month. For several days after, false labor complicated the situation. An acute pyelitis made its appearance 4 days before delivery, resulting in a birth 2 weeks premature. Figure 5, *a*, shows a ring of infarction and margin of membrane attachment. *b*, Shows edge of placental tissue. The placenta was of the typical circumvallata type, the edge of attachment of the membranes being about 2.5 centimeters from the true edge of the placenta. The baby weighed 6 pounds. The cord was attached to the center of the placenta.

Here is a case where the severe infection nearly terminated the otherwise normal pregnancy as evidenced by the false labor which followed. The reason is plain when we find the whole circumference of the placenta *as it was at the time* of the infection, infarcted. The only thing that saved the pregnancy was the persistent growth of the placental tissue, not from the injured edge but from the maternal surface within the edge which raised the infarcted edge plus the margin of membrane attachment away from the uterine wall resulting in the typical circumvallata formation.

CASE 12. Another case of severe bronchial infection was followed by a pinkish leucorrhœa which continued 2 weeks. The placenta showed an infarct which corresponded to 8 months at which time the infection and discharge occurred. Note also made in this case to look for infarct formation and it was there.

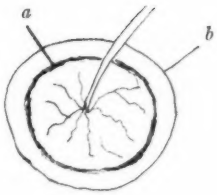

Fig. 5.

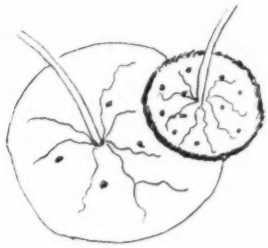

Fig. 6.

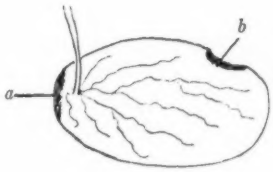

Fig. 7.

CASE 13. First seen in second stage of labor. Primipara. Last period October 13, 1920. Appendectomy in 1914. Frequent tonsillitis.

During first week of March, 1921, patient had influenzal attack. She was in bed 3 days at the end of which time she began to have a pinkish watery discharge. This was about 4½ months. This watery discharge, later colorless, was continuous until June 9 (7¾ months) when labor began. She was delivered of twins. First twin weighed 4 pounds, second 2 pounds. Second twin died in about 20 minutes. First twin died on third day of hæmorrhagic disease. It was not necessary to rupture the membranes of the second twin.

The difference in size of the twins was apparent from the size and condition of the placentas. The placenta feeding the second twin was only one-third of the total surface and this smaller placenta not only had its whole margin infarcted but was thoroughly studded with infarcts. There were a few small infarcts in the middle of the larger placenta.

In this case the inflammatory reaction of the infarct formation associated with the influenza attack undoubtedly resulted in the rupture of the membranes of the upper sac containing the second twin. This explains the continuous hydrorrhœa for 3 months and the absence of the sac of liquor when the second twin was born. The injury done to this one placenta undoubtedly accounts for the smaller size of the second twin.

This case is particularly illuminating in view of the case of twins recently reported by J. W. Williams of Baltimore (3) in which one twin was syphilitic and the other not. He cites this case as being consistent with Colles law, and to support this view suggests that the husband (not syphilitic) was the father of one twin and by a calling on the principle of superfœtation and Colles law he makes the syphilitic twin the child of a syphilitic lover.

The case I have cited offers a far more reasonable explanation. It is not so very uncommon for a woman with no signs of syphilis and a negative Wassermann to have a syphilitic baby. The spirochætæ in the child's liver have got to come from somewhere but if we conceive of a focus of spirochætæ somewhere in the mother which threw off a single embolus which landed in the placenta of one twin and not in the other and no more emboli followed, we have a much more reasonable explanation of the case than the one offered and one which is more consistent with other evidence. (See Case 18.) The infarct is a localized point of infection and as such has the same characteristics as the hæmatogenous boil. It is not the result of a bacteriæmia or septicæmia, otherwise the lesions would be multiple more often. Likewise if a breast abscess was due to a bacteriæmia, the abscess would be multiple more often.

In the case cited the placenta of one twin was damaged on the whole of its circumference with the result that it was prevented from enlarging to compensate for the needs of the growing fœtus. Its surface was also damaged, reducing the total available feeding surface of the placenta. Hence the discrepancy between the weights of the twins.

CASE 14. A primipara, last period occurred May 10, 1920. She had had frequent attacks of tonsillitis, quinsy sore throat, and bronchitis. Appendectomy was done 18 years ago. Besides definitely diseased tonsils, she had three devitalized teeth which the dentist declared were all right after X-ray examination. The films were not clear enough, however, to justify any diagnosis. About June 15, she had a slight bleeding spell.

At nearly every visit throughout her pregnancy she complained of her throat, coughing, headaches, pains in various places, feeling below par, dizziness, blurred vision and heartburn. January 20, her whole vagina became markedly inflamed, with itching, but very little discharge. January 28, she had an-

other bleeding spell. There was no rise of blood pressure throughout. In this case I made the note to look for a battledore placenta with another infarct to be situated about 5 inches from the base of the cord. Figure 7 is a diagram of placenta delivered. *a*, Represents the infarct which corresponds to the bleeding attack in the first month in June. *b*, represents the infarct which corresponds to the bleeding at the eighth month, in January. No other infarcts were visible to the eye. Normal delivery of 8-pound, 6-ounce baby.

It will be noted that in several of the previous cases, vaginal discharge with or without bleeding is associated with infarct formation. In this case the vagina became inflamed before the bleeding occurred, in others the bleeding came first. The onset of vaginal discharges (the discharge coming from the cervix associated with marked reddening of the vagina and itching) has been a more or less frequent occurrence in my practice. My present view of this symptom is that it results from the drainage of the septic serous discharge which originates in the early infarct formation. If the discharge is sufficient to dissect its way down between the chorion and the uterine mucosa to the cervix, it inflames the cervix and vagina. Bleeding may or may not accompany this symptom, dependent on its amount. One of the ward cases gave a history of slight bleeding about 2 weeks before delivery. When the placenta was born, an area of bleached out blood clot the size of one's hand was adherent to the membranes and extended from the edge of the placenta to the edge of the point of rupture of the membranes. In this case the external bleeding had been less than that which had been retained between the chorion and the uterine wall. The serous discharge which so frequently follows the frank bleeding in a case of threatened miscarriage is probably the expression of blood serum from the retained blood clot or from the inflamed area of beginning infarct.

Most of the above cases were collected during a period of 6 months and similar cases must be occurring in every obstetrician's practice. All that is required for their identification is careful history taking, note taking, and close observation of the placenta.

These cases not only demonstrate the significant relationship between the clinical events of the pregnancy and the findings in the placenta, but they also demonstrate the diversity of manifestations which are attendant upon infarct formation when it is appreciated that the infarct is infectious in origin.

The case which shows frank infection in the throat followed by uterine bleeding and this uterine bleeding is coincident with the formation of an infarct demonstrates a very clear sequence of events. It is apparent also that an infarct may form in the placenta without any outward manifestation, since the outward manifestation requires that the bleeding or serous discharge must be sufficient in quantity to dissect its way downward from the edge of the placenta between the chorion and the uterine wall to the cervix and vagina. Likewise chronic sepsis may cause infarcts without manifestations either in the mouth or vagina.

In these days when so much weight is given to laboratory and bacteriological evidence, it will be asked, Why not endeavor to find bacteria in these infarcts, and why have they not been found before?

A careful consideration of this feature of the problem will not only explain the last question but also will show that it is almost impossible of attainment.

In the first place it must be remembered that the acute infarct, that is, the one with active bacteria contained therein, is a red inflammatory affair. It can not be identified by the naked eye on the surface of the placenta. The white infarct which we see ordinarily is the end-result of this process which originated weeks and months previously. The white infarct does not represent present infection but past infection. It is similar to a healed scar. In order to identify the area of acute infection the placenta must be sectioned, stained and studied with the microscope. It seems to me that this explanation not only makes it clear why the etiological factor has not been discovered but also makes clear that this form of research is almost sure to be fruitless.

Placentas *in situ* are the only ones which offer a reasonably fertile field of examination.

It must be borne in mind that the bacteria arrive via the maternal blood vessels. It is probable that the evidence of infectious inflammation may be located principally in the uterine wall, even after the birth of the placenta. This feature may account for many of the foul lochias after careful asepsis at the time of delivery. Reference to the pathological report in the case of Cornell and Earle referred to in my other paper (2) and the second case described by Thompson in his article (4) on "Placenta Prævia" demonstrate by their findings that the infarct is the result of infection.

The history of the following case strongly suggests that an acute infarct may be torn open at the time of birth resulting in infection of both mother and baby.

CASE 15. A multipara was seen in consultation at the end of the second day after delivery. The mother had a temperature of 102 but did not seem severely ill. Nothing could be found in the case to account for her fever other than uterine sepsis.

The baby was moribund with very high temperature and respirations and a bright petechial rash over its whole body. The baby died next day.

Up to the time of delivery the mother had felt well. The onset of the fever in both mother and baby had come within the first 24 hours.

To explain such a case it is necessary that the source of infection which made its appearance in both mother and baby simultaneously must have been present before delivery. As there was no evidence of it previous to delivery, it is probable that the labor was the means of bringing it to light.

If we picture an acute infarct in the placenta, it is easy to see why both mother and baby became infected simultaneously. The baby received virulent bacteria into its blood stream during the contractions of the uterus in labor from the acutely inflamed area in the maternal vessels of the placental site. The detachment of the placenta following the birth resulted in the increased activity of the bacteria still in the uterine wall. The mother recovered because she had previously acquired an immunity to these bacteria which in all probability came from a tonsil which had been abscessed several times (quinsy) previous to her pregnancy. The baby had no such immunity and died.

The clinical events described in the above 15 cases demonstrates a clinical entity. The evidence is convincing when it is found that the shape of the placenta and the position of infarcts thereon can be prophesied with reasonable accuracy.

Case 11, as well as other cases in my practice, demonstrate the corollary to be true. This case had no untoward symptoms during her pregnancy until her attack of influenza. The cord was attached to the center of the placenta which shows that it had enlarged concentrically until the acute infection came.

Not only do these cases demonstrate the etiological factor in infarct formation but also they show that infarct formation is due to many different kinds of bacteria. The clinical sequence of events shows where treatment can best be directed in the prevention of the consequences which follow upon infection of the placental site.

In a subsequent paper I shall show evidence of the consequences of infection of the placenta, the relation of infarcts to toxæmia of pregnancy and the effects of chronic sepsis in the pregnant woman.

REFERENCES

PART I

1. MACKENZIE, SIR JAMES. The Future of Medicine.
2. TALBOT, J. E. A clinical study of the placenta. Surg., Gynec. & Obst., 1921, xxxii, 552–556.
3. WILLIAMS, J. W. The value of the Wassermann reaction in obstetrics, etc. Bull. Johns Hopkins Hosp., 1920, xxxi, 342.
4. THOMPSON, W. B. The treatment of placenta prævia etc. Bull. Johns Hopkins Hosp., 1921, xxxi, 228.

ILEOSTOMY FOLLOWING RESECTION OF THE COLON

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THAT deliberate ileostomy following resection of the large bowel is a rational and advantageous procedure seems to be logical, and in a series of such operations performed in the Mayo Clinic its practicability has been so marked as to warrant its adoption as a routine measure.

Resection of the right half of the colon is a technical procedure, safer, easier, and better from the standpoint of prognosis than the resection of the left half because (1) the mobility of the right half of the colon generally is such as to make it approachable without too much difficulty, and even in cases in which the mesentery is short the division of its outer leaf from the parietal peritoneum makes it possible to rotate the viscus toward the middle line and to reach its lymphatics and blood supply with comparative ease, and (2) the blood supply is more constant than in the left half, and, consequently, a resection up to a point of election can be made more readily without the fear of necrosis at the point of anastomosis. The blood supply of the ileum is of no moment and the continuity of the intestinal tract may be restored either by end-to-end or lateral anastomosis as the preference of the operator dictates. The former, has proved most acceptable to us. It is made with three layers of sutures, two of chromic catgut and one of silk. Should the lumen of the two ends of bowel be disproportionate the ileum is split opposite the border of the mesentery, as suggested by C. H. Mayo, and the anastomosis completed in layers. The mucosa is closed with chromic catgut, as in a Moynihan type of gastro-enterostomy, and the serous layers with one row of catgut and one of silk sutures. If the lumen is kept open by holding it between the thumb and index finger using these as a sort of removable obturator during the anastomosis, the danger of leaving too small an opening is avoided.

Because of the uncertainty of the blood supply of the left half of the colon as well as the technical obstacles encountered in attacking the splenic flexure and lower sigmoid, its removal is a formidable undertaking. Whether end-to-end, lateral anastomosis, or the Balfour method of using a rubber tube is resorted to in order to restore the continuity of its lumen, one hesitates to conclude without abdominal drainage because of the ever-present danger of leakage at the suture line. Fortunately, the few resulting fistulæ are usually of short duration and the local peritonitis which sometimes follows, rarely spreads.

Deliberate ileostomy in all resections of the colon reduces, to the minimum, distention from flatulence with resulting tension on the suture line and makes convalescence as uneventful as possible.

The advantages of an ileostomy as a "safety valve" and a ready drain to the proximal intestine are obvious. The idea of a safety valve against back-pressure from gas first was suggested by bringing out the appendix through a stab wound in the flank and later cutting it off to allow the escape of flatus; this is still practiced by many surgeons. The poisonous products of chronic, progressive, intestinal stasis, due to neoplastic obstruction, are such that the combined operation, that is, first, relieving the obstruction and then removing the offending growth, has forced itself into popularity. Whether one employs colostomy for a rectal or sigmoidal growth, or cæcostomy, as advocated by Stiles, drainage is established, and the second stage of the operation is rendered infinitely safer. We have not as yet put enterostomy to such use, but it seems to offer much the same advantage as following resection.

TECHNIQUE

We have found an adaptation of the Witzel method of gastrostomy simple and advantageous. It has seemed preferable to attach the loop of intestine with its tube into the lower angle of the incision which is usually

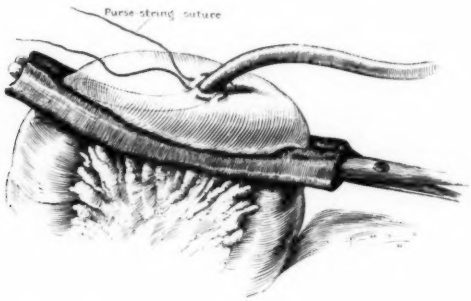

Fig. 1. Purse-string suture placed around the tube after its introduction into the bowel.

Fig. 2. Purse-string suture tied at the opening in the intestine, catheter buried by a row of interrupted sutures.

paramedial, rather than to make a new stab wound in one or the other of the iliac fossæ. Prolonged wound drainage from contamination is rare, and intestinal fistulæ have not been observed. A loop about 30 to 40 centimeters proximal to the line of anastomosis is caught up and held lightly in rubber-covered clamps after being stripped of its contents as dry as possible. In its center opposite the mesenteric border, a purse-string suture of silk is placed and through this an opening is made into the lumen of the bowel either with knife or cautery, as suggested by Long. A rubber catheter, with the end cut off and several fenestrations in the side, is inserted into the lumen, the purse string is tied and cut, and a new suture used around the base of the tube, making a second invagination. The tube is now laid along the long axis of the bowel and depressed into its wall, while the serous and muscular coats are drawn over it with the second silk suture, which has already been used around its base. The depression of the tube into the bowel wall for 2.5 to 5 centimeters insures against leakage and future fistula. It is well to fasten the tube into the bowel more securely by passing the last suture through the lumen of both. C. H. Mayo has sometimes employed the Coffey method of incising the bowel down to its mucosa and closing the outer coats over the tube (Figs. 1 and 2).

After completion of the enterostomy, the catheter is attached in the lower angle of the wound to the parietal peritoneum and the catheter is closed off with a clip, to be opened when occasion demands. We have found it a decided advantage to use a very small catheter since the fluid content of the bowel can be drained slowly if necessary; evacuation of gas is the primary object. We have used No. 8 and No. 12 catheters and have decided in favor of the smaller. The silk sutures through the lumen generally cut through readily and the catheter drops out of its own accord, usually about the twelfth day. In one case a larger tube stayed in 17 days. The tube may be opened at any time and drained into a bottle fastened in the dressing. The ease with which distention is thus prevented is in striking contrast to the persistent and less successful use of enemata (Fig. 3).

The surgical procedure should be supplemented by rigid adherence to the principle of giving nothing by mouth for several days after the operation. Fluids by hypodermoclysis amply supply the tissues with nourishment and are easily tolerated if given in moderate amounts. Morphine in adequate doses to combat pain and inhibit peristalsis is essential. The too ready acquiescence in hospitals to the patient's demands for food within the first week of convalescence is to be distinctly deprecated.

DISCUSSION

Nélaton, in 1858, successfully performed a deliberate ileostomy. However, years before, French surgeons had suggested some such practice in acute intestinal obstruction. Summers in 1920, in reviewing the history of en-

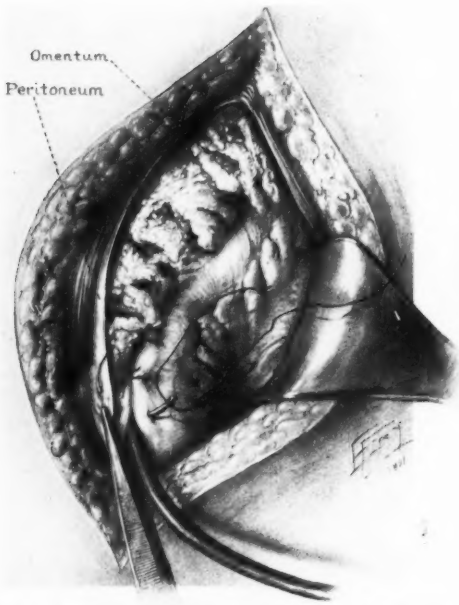

Fig. 3. Omentum brought down to cover the suture line. The needle passes through intestinal wall and peritoneum.

terostomy, credited to Renault its first employment in a case of strangulated hernia. Since then, various applications of the same principle have been devised and put into use as the indications were presented. Perhaps the most varied methods of technique have been evolved in attempting some form of jejunostomy for feeding in cases of malignant obstruction of the œsophagus or of growths in the stomach. The selection of the jejunum for enterostomy in cases of intestinal obstruction was first advocated by Victor Bonney, and later by Handley in contradistinction to the old and, up to that time, accepted theory that the first distended loop of intestine that presented itself should be opened and used.

While this procedure necessitates more manipulation and exposure than the simpler one of taking the first coil available, it drains the contents of Bonney's "segment of toxicity" more surely and successfully. The experimental work of a number investigators along the line of intestinal obstruction seems to bear out the contention that most of the fluid content of the intestinal tract is high in the small intestine.

BIBLIOGRAPHY

1. Balfour, D. C. The utility of the rubber tube in intestinal surgery. Surg., Gynec. & Obst., 1920, xxxi, 184–192.
2. Bonney, V. On postoperative paralytic obstruction of the intestine, with special reference to its treatment by jejunostomy. Arch. Middlesex Hosp., 1910, xxi, 39–44.
3. Coffey. Quoted by C. H. Mayo.
4. Handley, W. S. Ileus duplex (inflammatory enterocolic ileus). Lancet, 1915, i, 900–906.
5. Long, J. W. Enterostomy: a perfected technique. J. Am. M. Ass., 1917, lxviii, 833–835.
6. Mayo, C. H. Enterostomy and the use of the omentum in the prevention and healing of fistula. Ann. Surg., 1917, lxvi, 568–570.
7. Nélaton. Quoted by Summers.
8. Renault. Quoted by Summers.
9. Stiles, Sir H. The value of cæcostomy in the treatment of malignant disease of the colon. Brit. J. Surg., 1921, ix, 1–3.
10. Summers, J. E. Enterostomy in the treatment of acute intestinal obstruction. Surg., Gynec. & Obst., 1921, xxxii, 412–414.
11. Witzel. Quoted by C. H. Mayo.

TOTAL TRANSPOSITION OF VISCERA

By HENRY H. SHERK, M.D., F.A.C.S., Pasadena, California

As an anatomical curiosity *situs viscerum transposus* has always been of considerable interest to the anatomist and embryologist and instances of this abnormality have been observed and recorded since the time of Aristotle. To the clinician its recognition has more practical significance and is of extreme inportance from the standpoint of differential diagnosis.

Until (1) recent years most of the recorded cases had been found on the dissecting or post-mortem table. Now, however, the reverse is true and the majority are recognized during life by the clinician. The X-ray and the present day thorough and systematic routine examination are responsible for this.

Aristotle's writings (2) show that he had observed two instances of transposed organs in animals.

Petrius Servius (3), in 1615, recorded the first recognized human case as occurring in Rome, but Carl Beck (4) states that the earliest authentic case was reported in the time of Moliere, in the person of Marie de Medici, Queen of France.

Kuchenmeister (2), was the first to recognize the condition during life, based on examination by percussion and auscultation.

According to Sorge (1) the first X-ray record of this condition was made by Vehemeyer in 1897.

In 1865 Grueber (5) collected 79 cases from the literature, only 5 of which had been recognized during life.

Kuchenmeister (5), in 1888, collected 149 cases. By 1895 Pic (5) was able to collect 190 reported cases and in 1902 Arneil added, including his own, 44 more, 38 of which were recognized during life.

Karashima (2) in 1912 catalogued over 200 cases and gave what is probably the best historical résumé.

From the literature at my command I have been able to find 24 cases reported since 1912 and have received verbal reports of three unpublished cases (J. M. King, S. J. Mattison and H. A. Royster).

These figures cannot be accepted as accurate or complete for this anomaly has been reported under so many different titles and individual cases so often duplicated, in the published articles, that it is very difficult to sift them out.

A communication from the Mayo Clinic states a hurried search of their records shows 10 cases indexed since 1910. For the same period their registration was approximately 347,000.

Only two writers (6 and 7) have mentioned observing this condition in recruits during the late war. It would be interesting to have the figures of the findings, in this regard, of our draft and army examining boards.

By transposition of a viscus (*situs viscerum inversus* or *transversus*) is meant, not only that the organ is situated on the side of the body opposite to its normal location, but that it is also reversed as to its actual structural relationship, presenting, in comparison to the normal, as if seen in a mirror. For example, in transposition of the stomach the pylorus lies to the left and the cardia to the right, or, in transposition of the heart the relative position of the auricles, ventricles, and vessels is reversed.

When the viscus is not reversed in its abnormal location it is simply a displaced organ or in a condition of *situs perversus*.

Of course an acquired dextra cardia is never a true transposition of the organ.

Transposition of the vicera may be total or partial. The former is by far the more common. The latter is very infrequent. This is especially so of true congenital dextra cardia, as an isolated condition, of which Schrotter (8) says, "none, not combined with transposition of other viscera, have actually been confirmed by autopsy." Contrary to Schrotter's statement. Kriezer (8) in 1880 collected 14 reported instances of isolated congenital dextra cardia. Of these 14, how-

ever, only 5 were proven by post-mortem examination.

Total transposition of viscera is apparently twice as common in men as it is in women (5) and, when occurring in women, it has no effect on their childbearing ability (9).

Statistics on which to base the incidence of heredity could not be found in the lietrature, no cases having been recorded in which the condition occurred in parent and child. Brimblecombe (10) reports one instance of two cases in the same family, brother and sister.

Many theories, as to the determining factor in the causation of this condition, have been advanced—but none has been substantiated (5, 11 and 12).

The most plausible explanation (3) is the one that ascribes it to the changed position of the primary cardiac tube in early embryonal life, a reversal of its direction from a normal "S" to the reversed "2." This change (8) of position is probably caused by the reversal of the direction of the blood current which occurs if the usual relationship of the embryo to the primitive chorion is altered, so that its right, instead of its left, side is placed nearest its blood supply.

Von Baer (5) ascribes it to the changed relations of the embryo and the umbilical vesicle. Normally it lies on the left side of the vesicle, but if it lies on the right side transposition of the viscera occurs.

Rindfleisch (5) believes that a reversal of the spiral turning of the blood column is the causative factor.

Virchow (5) emphasizes the influence of the umbilical cord. In *situs transposus* it is wound spirally to the right, like a right-handed screw, the reverse of the normal which is to the left. This fact, if true, is of real practical importance.

Adami (12) quotes the monochorial twin theory, but puts little faith in it.

In his article (5), Arneil takes up most fully, the many theories that have been put forward by various writers.

A reversal of all of the ordinary physical signs occurs in total transposition of the viscera. The position of the apex beat, the cardiac dullness, and valvular sounds, the liver dullness, and the stomach tympany are all transposed and reversed. Also, in the male, the right testicle, in the majority of instances, hangs lower in the scrotum than does the left.

The reversed spiral twist in the umbilical cord may be present when the newborn infant is the subject of transposed viscera (5).

The X-ray findings simply show a picture as if the photographic plate, taken of a normal individual, were reversed.

Theoretically the speech center in the brain should be transposed (12 and 13). This is of special interest to the neurologist and the neuro-surgeon, in so far as it influences differential diagnosis. Correspondingly the subject should be left-handed, but this is an inconstant finding.

Electrocardiagraphic tracings sometimes show inverted waves (14 and 3)—but this also is not constant.

The transposed organs develop normally (3), and their abnormal position apparently has no effect upon their functioning or upon the general well-being of the subject of total transposition. They are normally able to undergo exertion without distress or undue fatigue and are not more than ordinarily susceptible to disease (15).

On the other hand the subject of partial transposition is generally deficient in stamina and suffers distress upon slight exertion (15).

Recognition of the abnormality is generally easy, except when associated with some pathological condition which might account for an acquired malposition of an organ.

It is surprising how many of the reported cases of transposition of the viscera remained unrecognized until the abnormality was disclosed at operation or autopsy.

Virchow (4) found transposed viscera, *post mortem*, in a case where, in spite of the observation of reversed ordinary physical signs, the clinician had stated that on auscultation the heart sounds were "heard with difficulty on account of the presence of bronchitis" and that the "spleen was very much enlarged."

One reported case had been examined five times for life insurance without the condition of transposed viscera being recognized (5).

Fig. 1. Roentgenogram of chest in author's case.

Fig. 2. Roentgenogram of abdomen in author's case.

Lane (6) reports a case of a soldier who had previously been examined several times and his abnormal condition passed unrecognized.

From a diagnostic standpoint the knowledge of the existence of a condition of *situs viscerum transposus* is of extreme importance in the presence of most of the inflammatory diseases and injuries of the chest and abdomen. In some its recognition is essential to a correct differentiation.

It is unnecessary to enumerate the many pathological conditions in which this has a bearing.

It is also unnecessary to emphasize the great importance of making a complete and thorough physical examination, with the possibility of this anomaly in mind, as a routine procedure in every case.

In newborn infants an umbilical cord with reversed twist should always suggest to the obstetrician the possibility of an associated visceral transposition. How seldom this is observed and how few babies are given more than a casual glance, to see if there is any gross external malformation, is only too well known.

Every individual, in whom transposed viscera are observed, should be fully advised as to the abnormality and be instructed, in case of illness or accident, so to inform the attending physician. This precaution may prevent an unnecessary operation or at least obviate inconvenient or duplicated incisions.

My own case, seen recently, is as follows:

Miss M. A., Norwegian servant girl, age 27, height 5 feet 6½ inches, weight 166 pounds net, consulted me October 14, 1921, on account of a gradually increasing amenorrhœa. Family history negative. Mother died of kidney disease. The patient was a large, hearty girl with bright complexion, who said she had had no illness since childhood, except an attack of influenza about 2 years ago. Her menses began at 13, irregular at first, 28-day type and lasting 3 to 4 days. She had little pain, which, when present, always occurred at the start. The flow was never free. She is not of nervous type. Does not overwork. Has no past sexual history, at least none could be elicited. Said her menses for past year had become less and less. Her last period had ceased 2 days previously. It had lasted less than 2 days, and the flow had not been more than a show. I examined her chest as a matter of routine and, in view of a possible pregnancy, to allow inspection of the breasts without

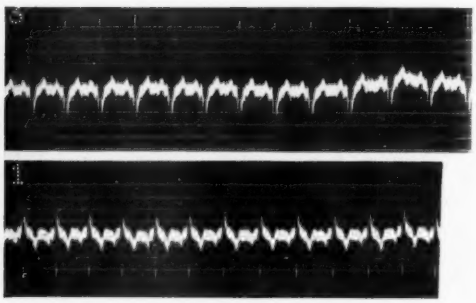

Fig. 3. Graph showing normal curve at top, *3*, reversion curve, in author's case, *1*, at bottom.

drawing attention to them. The breasts were well developed but negative. Auscultation of the usual heart area elicited rather faint heart sounds, though not more faint than often heard over normally located hearts. No apex impulse could be felt. On listening over the right chest the heart sounds were heard, loud and distinct, in the reversed position. The apex beat could be felt, in the right nipple line, exactly opposite to the normal position on the left. Percussion of the upper abdomen and lower chest showed tympany on the right and dullness on the left, the latter corresponding in size and outline to the ordinary liver dullness. There was no abdominal tenderness. The spleen and kidneys were not palpable. The external genitals were well developed and symmetrical. The introitus admitted two fingers freely. The cervix was small, hard, and conical and pointed downward and forward. The fundus was not definitely palpable, owing to the firm rigid abdominal muscles, but it certainly was not enlarged and was apparently retroverted. Nothing was discovered to suggest pregnancy. A second examination, made one month later, demonstrated an undersized, sharply anteflexed uterus with the body small in proportion to the cervix. The adnexa were not palpable.

The chest findings were corroborated by my colleague, Dr. J. D. Condit, who also made roentgenograms of the chest and abdomen. His report of the X-ray findings are as follows:

October 17, 1921. No. 3890. *Chest film* shows the heart with the apex on the right side, 3½ inches from the median line at a distance of 36 inches. The heart shadow is perfectly normal in size and shape, but reversed. The curve of the aorta comes at the normal relation, but to the right of the spine. The diaphragm shadow on the right side is lower than on the left side and shows the gas bubble in the upper pole of the stomach on the right. The relative height of the two sides of the diaphragm are normal. The chest is otherwise negative, except for a moderately normal anount of hilus thickening.

October 18, 1921. No. 3894. *Abdominal film*, barium meal and enema given simultaneously. The enema entered a normally placed rectum, passed through a small sigmoid, up the right side of the abdomen. The splenic flexure is placed on the right. The transverse colon passes across level with the second lumbar vertebra to a hepatic flexure normally related, but on the left side. The appendix did not fill. The ileocæcal valve is competent. The stomach is seen horizontally placed in the uppermost part of the abdomen, normal in size but transposed.

The entire contents of the chest and abdomen appear in the films as absolutely normal in size, shape, and relationship except for their transposition.

Electrocardiograms were taken by Dr. A. S. Granger whose report is here given:

There is a complete reversion of all the waves in lead 1 which is typical of a complete transposition of the heart, and only seen in that condition. Leads 2 and 3 replace each other, but no reversion of waves is seen. The mechanism of this is as follows:

Lead 1 is a symmetric one from the right arm to the left arm. A lead taken from the left arm to the right arm in a normal subject produces an inverted picture, consequently the left arm stands to the normal subject as does the right to the subject of transposition.

A lead from the right arm to the left leg (lead 2) in a normal subject is almost equivalent to a lead from the left arm to the left leg in the subject of transposition (lead 3). So in the letter, leads 2 and 3 replace each other, but neither shows reversion of the waves.

Besides the above characteristics, the cardiograms are entirely normal, and very typically show the condition of dextra-cardia present.

It is interesting to note that this patient had never been conscious, as some of these subjects are, of the heart sensations on the right side and, although examined physically several times when she had the influenza and once or twice at previous dates, the transposed position of her organs had never been recognized. She is right handed and there is no asymmetry of figure, spine, face, eyes, nose, throat, or neck.

SUMMARY

1. Total transposition of viscera is not so very infrequent.

2. Its standing has changed from that of an anatomical curiosity to a matter of clinical interest and importance.

3. It has no effect on the health of the individual.

4. Its recognition is extremely important in certain surgical emergencies.

BIBLIOGRAPHY

1. Jacobson. Am. J. Obst., 1917, lxxvi, 953.
2. Linebeck. J. Am. M. Ass., 1920, lxxv, 1775.
3. Hirschfelder. Diseases of Heart and Aorta. 3d ed., 560.
4. Beck. Ann. Surg., 1899, xxix, 593.
5. Arneil. Am. J. M. Sc., 1902, xxxiv, 885.
6. Lane. Boston M. & S. J., 1919, clxxxi, 641.
7. Parsons-Smith. Lancet, Lond., 1919, cxcvii, 1076.
8. Abbott. Modern Med. Osler, 1908, viii, 344.
9. Hartz. Med. Record, 1916, xl, 1027
10. Brimblecombe. Brit. M. J., 1920, p. 889.
11. Willius. Am. J. M. Sc., 1919, clvii, 485.
12. Adami. Principles of Pathology, 2d ed., 1910, i, 277.
13. Bell and Winn. M. J. Australia, 1920., Apr.
14. Owen. Heart, 1911, iii, 113.
15. Meyers. Proc. Roy. Soc. Med., London, 1921, xiv, 5.
16. Aich. Indian M. J., 1919, liv, 380.
17. Idem. 1920, lv, 59.
18. Toy and Ellis. J. Am. M. Ass., 1920, lxxiv, 322.
19. Billings. Philadelphia M. J., 1900, vi, 671.
20. Tyler. Am. J. Roentgenol, 1917, iv, 581.
21. Upson. Am. J. Roentgenol, 1921, viii, 385.
22. Case. Am. J. Roentgenol, 1916, iii, 332.
23. Kelly. Brit. J. S., 1920, viii, 137.
24. Horn. Ann. Surg., 1915, lxii, 424.
25. Gross. J. M. Research, 1918, xxxviii, 473.
26. Hupp. New York M. J., 1911, xclv, 422.
27. Pool. Ann. Surg., 1912, lvi, 940.
28. Cullen. Umbilicus and Its Diseases, 1916.
29. Jewell. Practice of Obstetrics, 1901, 87.
30. Parvin. Science and Art of Obstetrics, 1895, 134.
31. American Textbook of Obstetrics. 1895, 93.
32. Playfair. Midwifery, 1880, 109.

THE GERMICIDAL CHARACTER OF THE EMANATIONS FROM COLLOIDS OF CERTAIN SILVER SALTS

A Preliminary Report

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FOR some time our interest has been directed to improvements in the preparation of germicides suitable for application to mucous membranes. This interest was further stimulated by the studies of Colonel Hugh H. Young after his interesting report on mercurochrome 220. Knowing that metallic silver had the quality of preventing the growth of micro-organisms immediately around it, we hoped to secure a preparation which would exhibit this quality and at the same time be non-irritating and if possible non-staining. It would not be of value to record our failures, as our early efforts were without encouraging results.

When we began, however, with the colloidal preparations of silver, the results became more satisfactory, notably with the colloidal silver chloride. This proved to have a high germicidal value, was non-irritating and did not stain unless it was exposed to light before being washed. A series of laboratory tests encouraged us with the prospects of obtaining a better preparation than those now employed. We now make a preliminary report of our clinical observations and laboratory tests, with photographs of some of them. A detailed account of both clinical and laboratory experiments will be made later as our findings seem to open a new field for study and work.

The photographs of agar plates showing the various tests with silver wire, colloidal silver chloride, colloidal silver iodide, and other silver salts, on cultures of staphylococcus aureus show a fact which we think has not, hitherto, been observed; namely, *that colloidal silver preparations give off emanations which prevent the growth of micro-organisms immediately around the sealed glass tubes containing them.*

This fact became of still greater interest when we found that the colloidal silver chloride was non-irritating to mucous membranes even when employed in a strength sufficiently strong to be of greater germicidal value than carbolic acid.

The comparative harmlessness of this preparation was clearly shown by the fact

Fig. 1.

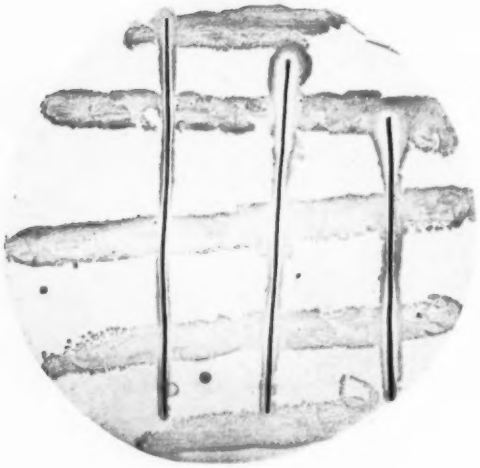


Fig. 2.

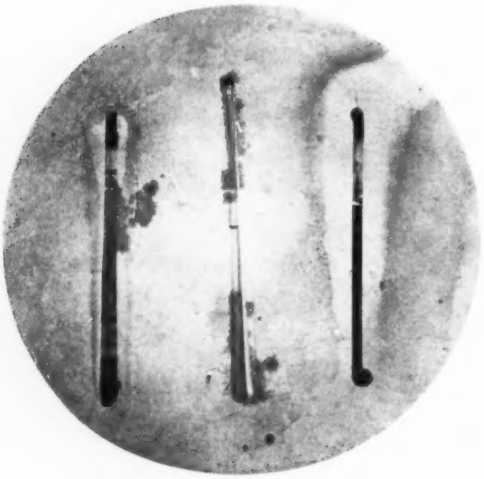


Fig. 3.

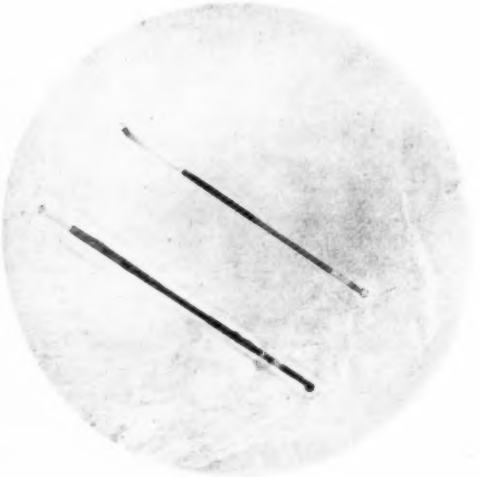


Fig. 4.

Fig. 1. Silver wire showing emanations destroying staphylococci around the wires. It was this phenomenon which led us to make the study of colloidal silver salts.

Fig. 2. *1* and *3*, silver iodide; definite emanations. *2*. Colloidal silver chloride, small in amount as shown at dark part. Small amount smeared inside tube. This shows emanations extending more widely from the part where there is a space in the tube.

Fig. 3. *1*. Colloidal silver iodide, emanations evident. *2*. Nitrate of silver, emanations negative. *3*. Colloidal silver chloride, emanations positive.

Fig. 4. *1*. Weak preparation; glass tube wrapped with 2 layers peritoneum; negative except emanation halo at burned end. *2*. Strong preparation, glass tube wrapped with two layers of peritoneum at one place, with 2 layers of cellosilk at the other. Definite penetration observed.

that spermatozoa could live and remain actively motile in it for 10 minutes or longer. The highly organized and delicate character of these cells would naturally show quickly the result of irritating or poisonous chemicals. When we found, therefore, that the laboratory reports of Dr. John Funke, professor of bacteriology at Emory University, showed

Fig. 5.

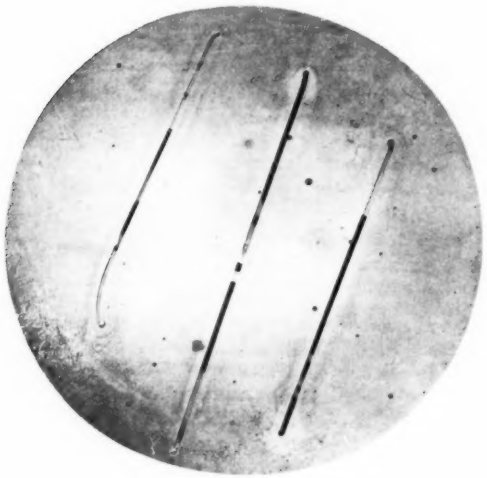
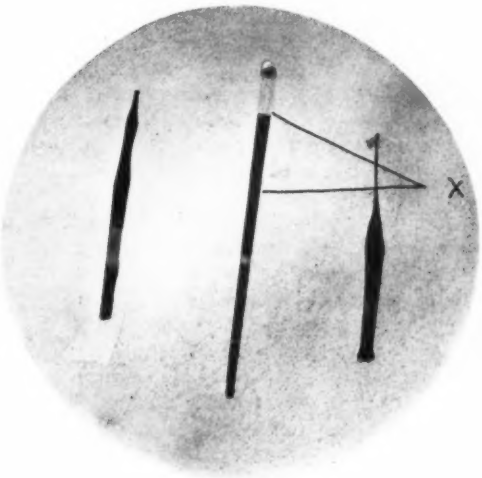


Fig. 6.



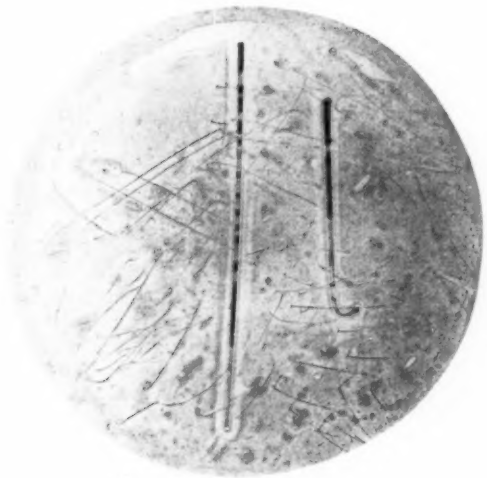


Fig. 7.

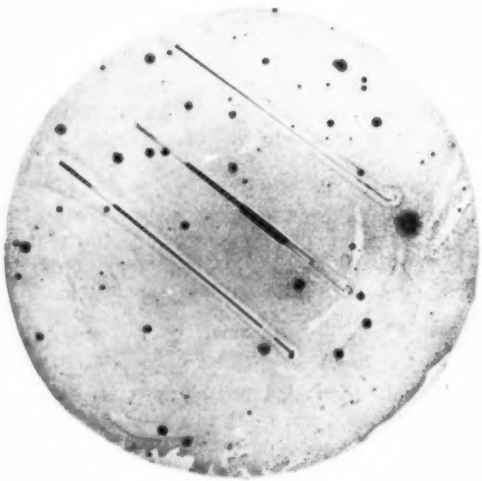


Fig. 8.

Fig. 5. All tubes colloidal silver chloride showing emanations, with wider sterile space at end of middle tube.

Fig. 6. *1.* Colloidal silver chloride in blue tube, thick glass, negative. *2.* Same preparation in thin clear glass, burned in flame except at point near clear end where emanation effect is apparent. *3.* Same colloidal silver chloride in thick yellow glass, negative.

Fig. 7. Colloidal silver chloride, positive emanations.

Fig. 8. *1.* Strong colloidal silver chloride in small tube; emanations positive; culture one week old and secondarily infected by photographer. *2.* Strong colloidal silver chloride in tube wrapped with four layers of peritoneum and covered with collodion; emanations slightly positive. *3.* Strong colloidal silver chloride in medium size tube, emanations positive.

that 5 per cent colloidal silver chloride, when diluted 400 times, killed the typhoid bacillus in 2½ minutes, and yet spermatozoa could live in the 1 per cent solution for 20 minutes or more, we felt that this preparation was remarkable. It clearly showed a selective action against micro-organisms and was not irritating to the body cells to which it was

applied. A 5 per cent solution of silver chloride diluted 4,000 times, mixed with an equal quantity of secretion from a foul mouth in which actively motile spirochætæ were observed, stopped the motility of the spirochætæ in 4 minutes. Many subsequent tests confirmed the early encouragement and now we have employed it in 300 patients with genito-urinary affections.

We were pleased to note in the July, 1921, number of the *American Journal of Urology*, an article by Dr. Ralph B. Cobb entitled "A New Advance in Silver Therapy," who reports very satisfactory results with colloidal silver chloride as prepared by Hille, the discoverer of argyrol. His observations of its clinical application to the mucous membranes of the urologic organs are in accord with our own work.

We sealed our colloidal silver chloride in the anterior urethra, using collodion over the meatus to prevent its escape, and it was without pain to the patient when so employed for 3 to 5 hours, nor was urination painful after the collodion was removed. The mildest remedy we have been able to use in this manner, heretofore, was argyrol, and yet it was not without discomfort and did not have anything like the germicidal value of the colloidal silver chloride, which Dr. Funke's report shows is several times stronger than carbolic acid. Neosilvol was non-irritating but lacked either germicidal qualities or penetration.

EMANATIONS

Colloidal silver chloride has the faculty of giving off emanations which apparently penetrate further into the tissues than any of the remedies we have been able, heretofore, to obtain. This fact is shown by both laboratory and clinical tests, for the clinical tests lead us to believe that we are able to destroy gonococci in the glands of Litre in the roof of the urethra when the solution itself could not enter them. The mucous membrane seems to be no more difficult for the emanations to penetrate than is glass, which the photographs of the agar plates show offers little obstruction.

We are unable to explain why some of the tests with colloidal silver choride show negative results as regards emanations, while others, quite similar, are positive. Our first thought was that there was a leak in the tubes and that this accounted for the emanation phenomena, but a careful study of the tubes and plates did not account for this. The thickness of the glass tubes accounted for some of the failures. Dilutions of the colloidal silver chloride accounted for others, while there still remains a series of negative results unaccounted for, except perhaps for the fact that traces of lead were in some of the capillary tubes or some of the sodium in the glass interfered with the emanations. We are duplicating our results with nonsol or Jena glass tubing to obviate the discrepancy. Tests were made with cellosilk, collodion and peritoneum from the intestines of pigs; all of these allowed emanations to pass through them (see photographs).

The reason that the colloidal preparation of silver gives off so much more of the emanation than the metallic silver is probably because of the enormous increase in the surface provided by the infinitely small subdivisions of the silver and the Brownian activity. It is not apparent why a 10 per cent solution of nitrate of silver should give off none or so little as not to influence the growth of bacteria on the agar plate; nor is it obvious why the colloidal chloride should give off more than the iodide. Equally puzzling was the fact that in the heated end of the tube, where some of the colloid is burned, there is frequently given off definitely larger amounts than from the part where only solution was in the tube, until we learned that Rutherford, in 1906, found that cocoanut charcoal absorbs radio-active emanations. Bancroft in his book on *Applied Colloid Chemistry*, 1921, in discussing this subject states that a slow current of air, at ordinary temperature, charged with emanations of radium, thorium, or actinium, is deprived of some or all of its emanations in passing through a tube filled with cocoanut charcoal. He further states in another experiment that Rutherford found that if a tube containing less than a gram of charcoal is connected to a vessel containing emanations from several milligrams of radium bromide, the charcoal will absorb the emanations in time. Further-

more, if powdered willemite be mixed with charcoal, the gradual absorption is shown by the increasing brilliancy or phosphorescence of the willemite. These facts explain why many of the burned ends of the glass tubes give off more emanations than the colloidal solution. The charred material in the burned ends evidently absorbs the silver emanations and then allows its escape with greater readiness than does the solution.

Empty spaces or bubbles also often destroy the germs for a greater distance than does the colloid. This phenomena is probably due to the wider play of the gaseous atoms. The Brownian movement so clearly seen with the ultramicroscope is only a visible demonstration of what is going on with infinitely smaller gaseous particles which are less obstructed by air than are the particles by the surrounding stabilizing solution.

Ehrenhaft[1] found that there is a much livelier motion in gases than in liquids, and his experiments led him to conclude that there is a motion in gases completely analogous to the Brownian movement.

The study of colloids, while most fascinating and intimately connected with much that goes on in and around us, is far too complicated and extensive to discuss in this connection. Many useful books have been written on colloids, their preparation and characteristics, and yet there remains much to learn about them. It is sufficient here to say that colloidal solutions of metallic substances or insoluble salts consist of minutely subdivided masses held in suspension by their Brownian motion.

CLINICAL USES

No irritating effect was noted when this preparation was given as an injection or instillation into the urethra, injected into the bladder, injected into the pelvis of the kidney for pyelitis; injected into fistulous tracts, abscess cavities or broken down buboes, applied to the conjunctiva, mucous membrane of the nose, throat or inner ear, antrum of Highmore, used as a wet dressing, etc.

Intravenous injections. Rabbits inoculated with fatal dose of staphylococcus aureus did not die if 1 cubic centimeter of a 1 per cent colloidal silver chloride solution was given intravenously 15 to 20 hours later. The control rabbits which received similar injections and not treated with colloidal silver chloride promptly died with multiple abscesses of the kidneys, heart, and other organs.

The rabbits were sometimes not disturbed by 2 cubic centimeters of the same solution given intravenously. Freshly made solutions were found to have greater germicidal value than the preparations several days or a week old.

Intraperitoneal injections. Five cubic centimeters diluted with an equal quantity of physiologic salt solution was given intraperitoneally and the rabbits lived, though they appeared sick on the day following the injection.

A series of experiments are now being made with tubercular and other infections than the staphylococcus aureus, and in due time we will report the results. The work is being done by Dr. Klugh in the laboratory of Drs. Bunce and Landham (who also made the agar plate tests) and by Dr. John Funke.

Silver chloride has the formula Ag. Cl. and is found free in its natural state as a substance known as horn silver; it can be prepared by mixing a solution of sodium chloride with a solution of silver nitrate and appears as a curdy, white precipitate, which darkens when exposed to light. It is decomposed by caustic alkalies to an oxide. It is soluble in ammonia water, thiosulphates and cyanides, and is insoluble in water and dilute acids. It melts at 500° F. and forms a translucent mass (horn silver). Finely subdivided in its colloidal form, its minute masses are negatively charged, moving to the positive pole (anionic). In a colloidal form it resembles diluted milk or whipped cream, according to the stabilizing solutions. The size of the infinitely small masses in the colloids vary according to its preparation, from those so small that they cannot be seen to the larger ones which are readily visible with a dark field microscope, as actively motile masses about the size of micro-organisms such as the staphylococcus or larger. Colloidal silver chloride is

[1] Sitzung. d. k. Akad. d. Wiss., Wien, 116, IIa, 1907.

miscible with fæces, urine, blood, gastric juices, and pus, and retains its colloidal characteristics for a few days to a week or longer according to conditions.

THEORY OF GERMICIDAL ACTION

The active bacteriotropic character of colloidal silver chloride leads us to wonder whether its activity is due to a chemical process or to the germicidal emanations. It seems difficult to conceive of its radiated atoms having sufficient size to combine in a chemical way with the micro-organisms in sufficient quantity to kill germs. Perhaps the emanation destroys the germ as does sunlight or emanations of radium. Subsequent studies may enable us to find how the germicidal action of the emanation affects its results. Colloidal disinfectants are adsorbed by the micro-organisms and thereby become concentrated on the surface of the organism and also penetrate the membrane which surrounds them.

Heidlberg, under a high power microscope, has observed colloidal solutions enter the gelatinous membrane covering the bacteria, after which the membrane swells and bursts. This observation, and also the fact that colloids possess high antigen power, as is evidenced by the fact that 1 gram of collodial iron will neutralize 50 diphtheria toxic units or 10 tetanus toxic units, may in a measure explain the germicidal action of the colloids.

COLLOIDAL BODIES IN THE BLOOD PROBABLY TAKEN UP BY THE LEUCOCYTES AND ENDOTHELIAL CELLS IN INFLAMED AREAS AFTER INTRAVENOUS INJECTIONS

The fact is well recognized in physiology that the leucocytes (phagocytes) "devouring cells" take up small foreign substances circulating in the blood stream. Our work so far leads us to believe that some of the colloidal bodies such as silver chloride are taken up by the phagocytes. These cells have also another mission, namely: to go to the points of inflammation, and thus they tend to concentrate the intravenous germicide where it is most needed. It would take us beyond the scope of our preliminary report to elaborate on this interesting phase of our experiments, concerning which we have some pertinent facts to discuss in detail later.

The surprising part of the work is that we have been able to carry on a series of successful experiments with a chemical so simple and one found in the pharmacopæia, the only thing necessary being to make it into a colloidal state.

An adequate supply of colloidal silver chloride is being sent to 200 of my medical friends to determine if their clinical results duplicate our own.

SUMMARY

1. Colloidal silver salts, notably the chloride, give off definite germicidal emanations. These emanations prevent the growth of micro-organisms on Petrie agar plates in the immediate area surrounding the sealed glass tubes containing the colloidal silver chloride.

2. A 1 per cent preparation has a germicidal value about equal to pure carbolic acid, yet when sealed in the anterior urethra for 5 hours is without pain, even with much stronger preparations. No irritation is produced when applied to mucous membranes such as bladder, nose, throat, sinuses, etc. Good results followed its application to abscess cavities, fistulous tracts, and also when used as a wet dressing for infected wounds, mastoid incisions, etc.

3. Spermatozoa will live in a 1 per cent preparation 20 minutes or longer. Being highly organized and delicate cells, this shows its relative harmlessness as a germicide.

4. Diluted 4,000 times, a 5 per cent preparation killed the spirochætæ from a foul mouth in four minutes.

5. Given intravenously to rabbits artificially infected with the staphylococcus aureus, 1 cubic centimeter of a 1 per cent preparation cured the rabbits, sterilizing their blood within 24 hours, while controls, untreated, died within 48 to 72 hours with multiple abscesses of the liver, kidney, heart, and other organs.

THE TEST OF LABOR[1]

Results in 100 Consecutive Deliveries and Factors Affecting Their Management

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Assistant Professor of Obstetrics and Gynecology, Rush Medical College; Attending Obstetrician, Cook County Hospital

MANY new obstetrical procedures are being advocated today, some to be employed almost as routine measures, each one as a panacea for certain obstetrical difficulties. Prophylactic forceps, routine podalic version and extraction, or mechanical induction of labor at a definite date, determined by estimation of fœtal age, are some such already prominent, and prophylactic cæsarean section may not be long delayed. Such titles in themselves, at times, tend to dissolve criticism. Who of us but is interested in prophylaxis? All of these measures have such earnest and able sponsors that we are thereby committed to impartial weighing of their merits, even though some must finally be ruled out perforce as being mutually incompatible. We here are concerned only, however, in trying to determine the value of methods long used, to enable us to compare the value of such older practice with the newer methods.

Does not the story of "Aladdin and his wonderful lamp" offer an allegory for us? There the cry "new lamps for old" was effective and though doubtless many benefited who gave their old possessions, still the fact remains that Aladdin's lamp was exchanged for one incomparably inferior though new. The new always attracts, sometimes from mere desire of change, sometimes from the hope that it represents better things. But now, as in Aladdin's days, real worth may perhaps be recognized only with effort and analysis.

What are the best methods to use to improve obstetrical results? To answer this question we may paraphrase the familiar old saying: "the proof of the pudding is the eating" as follows, "the proof of the method is the result." Therefore we must compare those results that are variously obtained.

Labor is the active efforts of the pregnant uterus to rid itself of the viable products of conception by childbirth. The Art (or practical application of our knowledge), of obstetrics largely concerns labor. One definite method of procedure, that we call "the test of labor," utilizes the natural forces and mechanism in labor to their full measure.

This paper is an analysis of results from this method in 100 consecutive cases. It is our belief that the test of labor is obtained only by allowing labor to proceed until childbirth either results spontaneously, or from operative assistance that reinforces the natural powers, so that birth is through the natural passages. Certain women should not be submitted to this test of labor. Definite pelvic obstructions, whether from marked contraction of the bony structure, massive scar tissue, or from the encroachment of tumors upon otherwise normal passages, usually present absolute indications for primary abdominal section. Also, when factors endangering life suddenly arise before, or very early in labor, that, for their successful treatment, necessitate prompt positive emptying of the uterus, the primary indication is for operative interference by a method appropriate to the emergency conditions, whether by abdominal section, vaginal cæsarean or *accouchement forcé*. But such cases, not suitable to the test of labor, are relatively very few.

The test of labor should eliminate subsequent consideration of abdominal section as a method of delivery for the given pregnancy, for we believe that a few hours only of first-stage labor is futile in determining the answer as to whether successful delivery through the natural passages is possible. Miscalled tests of labor are tried at times in cases of relative disproportion, although such procrastination may increase the risks of abdominal section when done, while it does not eliminate the possibility of successful pelvic delivery, because not carried far enough. A true test of labor, even in border-line cases, will certainly be followed by the delivery of living and

[1] Read before the Chicago Gynecological Society, December 16, 1921 (see p. 120).

uninjured babies in many instances, often operative but spontaneous in not a few. Even in the percentage where the fœtus of a particular labor does not survive, benefit to subsequent pregnancies may and generally does ensue, because fuller knowledge will indicate definitely that certain of these mothers are entitled to primary abdominal section, and that others will have successful natural labor subsequently, even though needing operative assistance in the present one. A completed test of labor, therefore, practically eliminates thereafter the borderline type of case.

In the series of 100 consecutive patients, who had the test of labor, were 54 primipara and 46 multipara, 99 longitudinal presentations, 97 of which were vertex and 2 breech presenting parts, and 1 twin pregnancy, the first vertex, the second breech. Of 98 vertex cases, 69 were anterior positions, 2 transverse, and 27 were posterior. Fifty-one babies, including the uniovular twins, were male, 50 were female. Of these, 3 were born dead, and 1 died in 18 hours following operative delivery; 1 spontaneous delivery died in 3 days after birth from inhalation pneumonia.

The largest male baby of operative delivery weighed 9½ pounds, of spontaneous delivery 9¼ pounds.

The largest female baby of operative delivery weighed 8¾ pounds, of spontaneous delivery 8½ pounds.

The spontaneous deliveries varied from 21 hours, a primipara, to 1½ hours, a para-V.

The following table gives the percentage of occurrence in the primiparæ, multiparæ, and in combination:—

	54 Primipara	46 Multipara	100 Total
High interference	28	07	18
Mid-interference	09	02	06
Low interference { Forceps	35	19	28
Low interference { Extraction	00	04	02
Spontaneous	28	68	46
Total	100	100	100
Anterior position	60	84	70
Cord { About neck	22	30	25
Cord { Low-lying	06	00	03
Dry labor	35	24	30
Oligohydramnios	22	09	16
Intrapartum hæmorrhage	22	25	23
Beginning maternal exhaustion	26	07	17
Threatened rupture of uterus	07	00	04
Constriction ring	06	00	03
Fœtal exhaustion	28	15	22
Episiotomy	43	15	31

Indications for operation were usually not urgent: 16 had inertia uteri, 1 was a first of twins, 4 were frail women, 1 of whom had acute diphtheria; 7 had exhaustion of the fœtus, 1 from cord about the neck, 2 from dry labor or oligohydramnios, 3 from combinations of these, and 1 from cause not ascertained. Spontaneous birth gave 5 babies needing resuscitation from 2 breech, 1 dry labor, 1 oligohydramnios, 1 low placental implantation cases.

There were 8 slight degrees of pelvic contraction, all in primiparæ, 2 flat in mid-forceps cases, 4 flat in low forceps cases, and 1 flat and 1 justominor in spontaneous birth. Of the 29 cases where the funis was involved, 1 had cord thrice about the neck, 4 twice, 1 had cord about neck and arm, 1 had a recent knot tightened during birth, in 3 the cord was low-lying and discovered only after operation was well started. The 5 premature labors include 1 (mid-forceps) in a primipara, and 1 with breech presentation in a multipara. Five very rapid labors occurred, all in multiparæ, 2 of these being apparently due to the only cases of polyhydramnios in the 100 cases. No serious cases of intrapartum hæmorrhage occurred. Eleven were third-stage in onset, and 5 second-stage; of the latter 4 were from low placental inplantation in multiparæ with spontaneous birth, 1 low placental implantation was found following a high operative delivery. Episiotomies were usually done, only after evidence that tearing was otherwise unavoidable. These combined with the number of second-degree tears represent about 50 per cent of the total number of patients. The cardiac conditions noted did not appear grave until labor had been in progress for a considerable time. No marked sepsis occurred except one abrupt onset of mastitis on the seventh day, following spontaneous delivery after short labor in a primipara with serious antecedent toxæmia. Both breasts were involved in all their quadrants and the infection apparently was due to abnormally low resistance, the invasion taking place through the milk ducts. Two cases with fever late in the puerperium were cured by quinine treatment, malaria-plasmodia being found. There was 1 manual re-

FACTORS IN HIGH AND MID-OPERATIONS

Para	Number hours to operate	Labor overdue	Position	Station	Dry labor	Oligohydramnios	Induction {B—Bag G—Gauze}	Delay and lack of progress	Beginning maternal exhaustion	Threatened rupture of uterus	Constriction ring	Cord about neck	Cord low-lying	Fœtal exhaustion	Result to baby	Method of delivery
I	24	—	P	High	—	x	—	x	—	x	x	—	x	—	Dead	H. F.* ff. by V. & M. Ex.
I	10½	—	P	High	x	—	—	x	x Cardiac Dila.	—	x	—	—	—	Dead	H. F. ff. by Craniotomy
I	24½	—	P	High	x	—	—	x	x	—	—	x	x	—	Dead	H. F.
I	16	x	P	High	—	x	B	x	—	x	—	—	—	x	Died 18 hrs.	H. F.
I	12	—	P	High	—	x	B	x	—	—	x	—	x	x	Living	H. F.
II	25	—	P	High	—	x	B	x	x	—	x	—	—	x	Living	H. F.
I	8	—	P	High	—	x	B	x	—	—	x	—	—	x	Living	H. F.
I	19	—	P	High	—	—	—	x	x	—	Sharp Prom. x	—	—	x	Living	H. F.
II	16½	—	P	High	x	—	—	x	x	—	—	—	—	—	Living	H. F.
II	24	x	P	High	—	—	G	x	x Myocard.	—	—	—	—	—	Living	H. F.
I	20½	x	P	High	—	—	—	x	x	—	—	—	—	x	Living	H. F.
I	9½	—	Transverse	High	—	—	—	x	x	—	x	—	—	x	Living	H. F.
I	6	—	P	High	—	—	G	x	—	—	—	—	—	x	Living	H. F.
I	35	—	A	High	—	x	—	x	—	—	—	x	—	—	Living	H. F.
I	9	—	A	High	x	—	—	x	x	—	—	—	—	—	Living	H. F.
I	9	—	A	High	x	—	—	x	x	x	—	x	—	x	Living	H. F.
II	7½	—	A	High	—	—	B	x	Cardiac case	—	—	—	—	—	Living	H. F.
I	25	—	A	High	—	—	B	x	—	—	—	—	—	—	Living	H. F.
I	23½	—	A	Mid	x	—	—	x	x	x	—	—	—	—	Living	Mid-Forceps
I	15	—	A	Mid	x	—	—	x	x	—	—	—	—	x	Living	Mid-Forceps
I	10	—	A	Mid	—	—	—	x	x	—	—	x	—	x	Living	Mid-Forceps
II	6	—	P	Mid	—	—	—	x	x Cardiac	—	—	—	—	—	Living	Mid-Forceps
I	17	—	P	Mid	—	x	—	x	—	—	—	—	—	—	Living	Mid-Forceps
I	11	—	P	Mid	—	x	—	x	x	—	—	—	—	—	Living	Mid-Forceps

*H. F.=high forceps.

moval of the placenta done. The diphtheria patient had labor apparently started from administration of antitoxin, as there were no signs previous to it, while promptly thereafter vigorous uterine contractions began.

High forceps usually involve intra-uterine application of the blades and admittedly make very serious demands upon both doctor and patients. They are, therefore, condemned by some *in toto*. However, it seems to us that, where the delay is first-stage essentially, that is, due to cervix and lower uterine segments not being dilated, rather than second-stage, that is, where there is bony obstruction or obstruction from tumor prolapse, there is a marked differentiation possible in the matter of procedure. We should bear in mind that in every dry labor or oligohydramnios that unaidedly completes the first-stage of labor, these uterine parts have been dilated by pressure of the fœtal head, hence high-forceps assistance in such like conditions may often bring the same results without damage to the mother or child, if such instruments are skillfully used without undue speed or excessive force.

Certain of the tabulated cases could probably profitably have had earlier induction of labor, so benefiting from a smaller baby. It is of interest that the two severest

FACTORS IN LOW OPERATIVE AND SPONTANEOUS DELIVERIES

	PRIMIPARA		MULTIPARA		
	Low operation	Spontaneous	Low operation	Breech	Spontaneous
Number	19	15	9	3	31
Presentation	Ver	Ver	oV	3Br.	Ver
Position, posterior	5	4	0	0	4
Position, anterior	14	11	9	0	27
Cord about neck	4	4	5	1	6
Cord low lying	0	0	0	0	0
Dry labor	7	5	0	0	11
Oligohydramnios	2	3	3	0	0
Intrapartum hæmorrhage	5	1	2	0	7
Beginning maternal exhaustion	4	0	1	Cardiac collapse	0
Fœtal exhaustion	7	1	2	2	2
Episiotomy	13	1	4	0	2

cases therein with babies born dead have each since had spontaneous birth of normal babies—as have also several others listed in these high operations on primiparæ.

Consider that these cases all were in labor for a considerable period before operative interference, and that the only early evidence of deviation from normal in most of them was lack of progress. That makes it plain that the recognition of lack of progress, and its underlying causes, is of very great importance, which is not always appreciated. Certainly obscure conditions, not to be diagnosed early, can hardly be available for preliminary vital decisions as to methods of delivery.

We believe that while other methods than high forceps might still have been used in most of these cases when finally the obscure conditions were recognized, maternal mortality would not thereby have been lessened and that indeed we might have had a marked increase in it, by any other method of delivery which did not, *pari passu*, increase markedly the fœtal mortality.

CONCLUSIONS

1. No patient should be submitted to the test of labor in whom are either obvious obstacles to pelvic delivery or emergency conditions necessitating speedy emptying of the uterus.

2. The test of labor, therefore, primarily concerns cases in which spontaneous birth is hoped for, in the present and subsequent pregnancies. Operative delivery, when spontaneous birth does not ensue, should be by the pelvic route.

3. Primiparity, posterior positions, dry labors, oligohydramnios, and constriction rings of the uterus, increase the percentage requiring operative delivery, particularly when they occur in combination. Constriction rings occur practically only where oligohydramnios or dry labor are present.

4. Lack of progress for a considerable time is the predominant indication for interference. Occurring alone the determination of the moment for such interference demands careful consideration and wise judgment.

5. Beginning maternal or fœtal exhaustion, or threatened rupture of the uterus, developing with lack of progress, present indications for immediate interference. Any lack of progress, however, demands prompt, careful analysis to determine its probable cause with particular watchfulness for the development of such subsequent urgent indications for interference.

6. Following a given test of labor, subsequent pregnancies should have an improved prognosis.

7. Forceps will always be the most used method of terminating non-spontaneous labors; therefore, the technique of forceps operation is of paramount importance, and should receive greatly increased attention in obstetrical teaching.

ROENTGENOGRAPHY IN OBSTETRICS[1]

By D. A. HORNER, M.D., Chicago
From the Chicago Lying-in Hospital

SINCE the beginning of my work in this field 2 years ago, several articles pertaining to the use of the X-ray in obstetrics have appeared in American literature. Heretofore, very little has been written, but it is safe to say that the near future will bring out much more, depending upon the readiness of the roentgenologist and the obstetrician to accede to the harmlessness of exposing the pregnant woman.

As an advance in obstetrics, roentgenography ranks with other comparatively recent discoveries, such as twilight sleep, pituitrin, rectal examinations in labor, the head stethescope and the low type of cæsarean section.

HISTORICAL

The surgeon and internist readily recognized the value of the X-ray in their own fields. Shortly after its acceptance by them, it became malpractice not to use it in various conditions. Today one wonders how they did without it for so long.

The obstetrician feared its action on the pregnant woman and the unborn babe. Only the bolder ones experimented with it half-heartedly. Superstition attributed to the apparatus all pathological conditions following its use. The timidity of the laity and the profession is even to this day quite pronounced.

The steps leading up to present-day use are few. In 1899 Mullerheim roentgenographed women with partial success. Before that, only fractional portions of the mature fœtus could be demonstrated. Thereafter, plates of the fœtus *in utero* were looked upon as roentgenographic curiosities. In 1913, Sjorgren of Sweden differentiated for the first time between intra-uterine and extra-uterine pregnancy. In 1912, O'Donnell stated that the position of the fœtus could be clearly determined from the fourth month. I shall consider this statement later. DeLee then in discussion fully outlined the limitations and possibilities of the X-ray. He emphasized its uselessness in determining the possibility of mechanical difficulties in labor except in gross deformities. In 1915, Judd showed a case of twins. In 1918, MacKenzie attempted measurements of the pelvis by this means and was partially successful. In the same year came the revolutionary work of Warnekros of Berlin on the fœtus *in utero*. Today, in all parts of the civilized world extensive roentgenographic investigations during pregnancy are being made.

PREGNANCY

By the ordinary means of roentgenography, I am convinced that it is impossible to show a fœtus *in utero* before there is sufficient ossification to form a skeleton. Both Hess and Adair arranged schedules for the appearance of the ossification centers in specimens taken from jars; but showing these centers in the fœtus *in utero* is another matter. Their density at 90 days is not great enough to show through abdominal and uterine walls as well as liquor amnii, until long after they can be demonstrated outside of the body. It is only after they are well developed and widespread that they can be attributed to fœtal bones and not to calculi, enteroliths or phleboliths, etc. At 120 days, or 4 months, only that part of the fœtus over the inlet is demonstrable, the rest being obscured in liquor amnii, muscle, and maternal bones.

O'Donnell, Albert Weil, and others claim positive diagnoses are possible at 4 months. Edling, as early as 1910, and Fornero, as late as last May, stated that at 3 months not only pregnancy but also presentation and *position* can be determined.

Unless these investigators have a refinement of technique beyond that of most present-day roentgenologists, their researches must be considered faulty. My own efforts satisfy me that the fœtus cannot definitely be diagnosed as such before quickening. After this it becomes a comparatively easy matter in women of average weight.

[1] Read before Chicago Gynecological Society, December 16, 1921. (For discussion see page 120.)

It was hoped that pneumoperitoneum would reveal something of value in the early months. It reveals nothing that the educated finger cannot tell us. Its place in obstetrics is associated with the recognition of accompanying tumors, adhesions, and ectopic pregnancy.

"Position" as stated by O'Donnell and Fornero must not be confused with "presentation." I have found in my study that the X-ray plate shows the size and clearness of the fœtus required to demonstrate "position." The collection of Warnekros is also called to your attention for a similar purpose. General practitioners sending patients to hospitals for deliveries often have no conception of the presentation and position in their cases. They depend for this information on the internes of the hospital even after having had their patients under observation for months. For such as these I would suggest at least one exposure before the onset of labor.

FŒTOMETRY

After demonstrating the fœtus, fœtometry is the next advance. The work of Hess is of value in establishing the age of the expelled fœtus. But there are occasions when the age of the fœtus *in utero* as well as its actual size is required. We attempt to measure its length and head diameters. The induction of premature labor and the prevention of over-term pregnancy can now be undertaken with a greater degree of certainty than heretofore. The dates of last menstruation, of conception, and of quickening, if available, are important in making decisions of pregnancy duration from plates.

It is not unusual for even the best obstetricians to be from 1 to 3 pounds off in their estimates of the size of the fœtus.

When abdominal palpation and mensuration are not satisfactory and dates are unreliable, roentgenographic interpretation can be depended on.

Six of my series were shown to be too large to navigate the passages successfully. Cæsarean sections were performed in these cases. Six mothers were thereby spared useless, difficult labors with extensive damage, and six babies who otherwise might not have survived are living today. In none of these cases was the fœtus over-term. Pelvic measurements were close to normal.

At this point I may state that in not uncommon instances, cæsarean sections have been performed bringing forth deformed babies or monsters, thereby assuming an unnecessary risk for the mother. Several obstetricians insist on routine roentgenography before cæsarean section to view beforehand what the uterus contains; others are still fearful.

Objection has been raised to the use of the X-ray just prior to cæsarean section on the basis of its devitalizing power, comparing its effects on the peritoneum with that of radium. Fatal peritonitis has followed the administration of radium in uterine conditions. The shortness of the exposure to the X-ray as compared with radium, renders this procedure harmless. My experimentation on pregnant women shortly before cæsarean section has not been stopped by the faintest suspicion of peritoneal involvement. It follows that, given clean cases, peritonitis is still attributable to a slip in surgical technique and not to the X-ray. The endothelium of the knee joint is more sensitive than the peritoneum, yet the surgeon does not hesitate to employ it there.

PELVIMETRY

When DeLee (1912) stated that one could recognize *gross* deformities, distortions, and narrowing of the "inlet," practically nothing had been done to measure the pelvis with a view to ascertaining its *finer* defects. The recognition of gross deformities as related to childbirth is quite an accomplishment in itself, and is much to the credit of the X-ray. We often, in our crude external pelvimetry, take too much for granted when the measurements are normal. Difficulties encountered later, when the patient is advanced in labor are often unlooked for. Every obstetrician has encountered such cases. When discovered it is too late to do an abdominal operation. The expert obstetrician, it is true, would not be confronted with such problems as one not so skilled. The former realizes the value of the test of labor, the latter does not.

In many instances one can say that a certain head will successfully come through a given pelvis, but in a number (far greater than we admit), it is impossible to state so, positively. The importance of *knowing* definitely is more evident with each labor terminating with difficult high forceps and fœtal death. The conjugata vera and the bispinous measurements can be taken with the fingers or special instruments with a fair degree of accuracy. But we are not all proficient in ascertaining these cardinal internal measurements.

Many defects overlooked in external and internal examinations, e. g., marked contractions of the inlet, sacro-iliac disease, effects of old fractures, exostoses, callus formation following previous pubiotomy, etc., can be perceived at a glance.

The recognition of the finer faults can only be made by the X-ray if at all; and then only after much experience in interpreting the plates taken. The following-up of such cases clinically is essential to successful interpretation and is possible only in hospitals with large maternity departments; comparison in many cases being required. Collaboration of roentgenologist and obstetrician is of prime importance.

There are possibilities of error in interpreting the size of the inlet because of the varying degrees of tilting of the different pelves. If all pelvic inlets had the same angle of inclination to the horizontal, it would be an easy matter to estimate their measurements.

Higher mathematics have been employed in roentgenographic pelvimetry. The methods and calculations are too complicated for general use. But recently, I experimented with Chamberlain and Newell's stereoscopic method. The results obtained are excellent. Only a few simple geometric principles are involved in the solution of any or all the diameters.

Stereoscopic views at or just before term give a much clearer picture of the relation of the head to the maternal pelvis, and the probabilities of difficulty in engagement.

The measurements in cases of ankylosis of the sacro-iliac joint and the assimilation types of pelves may be perfectly normal, yet abnormalities in the mechanism of labor occur. The X-ray is the only antemortem method of positively diagnosing these types, laparotomy not excepted.

Recently a nurse asked that her pelvis be roentgenographed as she was about to marry. This unusual request was from a trained individual who realized the gravity of childbirth. It makes one wonder why more such requests are not made. Even the ignorant, pregnant woman desires to know if "everything will be all right." Physical examinations for disease are required in several states before marriage, but seldom is the woman's fitness to deliver a normal-sized child questioned. The grossest deformities of her pelvis are not considered a detriment, even though her first labor will be fatal to her and her child, if not conducted by one specially prepared for all complications. A good share of the 17,000 annual deaths in this country resulting from labor could be spared if there existed a more general application of the X-ray for investigations of this sort.

With maternal conservation the watchword of the day, the above paragraph is more than speculative.

NEWBORN

Infants are subject to trauma in the birth process. Without the X-ray many of these injuries are overlooked. With it, early diagnoses can be made and proper treatment immediately instituted. Fractures and dislocations of any of the long bones are not uncommon. Skull fractures are more difficult to locate.

We have also demonstrated dextrocardia, pyloric stenosis, enlarged thymus, effusions in the thorax, atelectasis, and tumors.

OTHER USES

We have also demonstrated the extent of upward and lateral displacement of the fœtal head during the use of hydrostatic bags, thereby showing the ease with which a cord may prolapse following the expulsion of the bag. We have also shown a transverse presentation accidentally produced in the introduction of a metreurynter.

There are many others uses to which the X-ray can be applied. Pseudocyesis, obscure presentations, deflexion attitudes, and questionable multiplicities are easily settled. Associated surgical conditions in pregnancy and the puerperium are not uncommon. Calculi of the urinary and biliary tracts have been located and surgically treated without interruption of pregnancy.

The presence of foreign bodies in the generative organs is easily determined. This statement, when applied to obstetrics, may appear rather peculiar. But catheters, bougies, and pessaries used in various minor operative procedures, have been lost or forgotten. A Y-shaped, brass pessary was recently located in a freshly delivered placenta by one of my colleagues. The patient, when questioned concerning it, denied all knowledge of its existence. Unfortunately in this instance no roentgenograph was taken. Discovery might have created a knotty problem. A similar instrument was located in the fundus uteri of an amenorrhœic stout woman whose physician had inserted it as a cure for sterility. A roentgenograph revealed it although she had supposed it removed years ago. Its removal was followed by pregnancy, which might never have occurred without the roentgenograph.

In my review of the literature I have not come across the utilization of the X-ray in the diagnosis of intra-uterine death. However, during the past year in 3 cases where fœtal death was suspected. X-ray revealed overriding of the skull bones with asymmetry of the head. This to me indicated fœtal death and the subsequent clinical course bore out my belief. Therefore, overriding of the skull bones with cephalic asymmetry are signs of fœtal death and are the only positive signs of intra-uterine death.

EFFECT ON MOTHER

No appreciable effect on the mother has been noted in the past 2 years. I have been told that I have unknowingly produced sterility. Even temporary sterility is out of the question, as the exposures in most instances have been shorter than those required for gastro-intestinal roentgenography. Fraenkel's experiments on prolific lower animals showed that the ovaries will stand a great deal before losing their function.

Not a single instance of X-ray burn has occurred.

EFFECT ON FŒTUS AND NEWBORN

Thousands of women are continually subjected to the X-ray for other than obstetrical conditions before and during the early months of pregnancy, without effect on the ovum. I do not mean to imply that the X-ray cannot kill an ovum, but refer to the use of the apparatus in diagnosis. The modern abortionist uses it to kill.

Most of my cases were fearful of injury to the babe.

Werner recently mentioned three children of mothers exposed during pregnancy to X-rays 5 years previously. The children were normal in all characteristics except height and weight. How and why he attributes these deficiencies to the X-ray exposures of 5 years previous is not clearly seen. The same can be said of Aschenheim's imbecile, the child of a woman treated for uterine myomata; conception occurring between exposures. Stettner now adds two instances in which the eyes, ears, and genitals were deformed. Deficient growth, inferior mentality (as in Aschenheim's case) and disturbed co-ordination also existed.

Nuernberger's experiments on lower animals showed the spermatozoa, ova, and offspring normal, following exposures of minutes.

Attributing to the X-ray isolated cases of malnutrition or imbecility is as ridiculous as to ascribe the occurrence of club foot in a child to the fact that its mother had sprained her ankle during pregnancy. Many women who have never come near the apparatus have brought into the world similar children and even worse monstrosities.

Exposures in my cases were limited to seconds while Aschenheim's and others required minutes for their treatments. We wish to go on record as having given one patient twelve exposures totaling over 3,000 milliampere seconds during the last 2½ months of her pregnancy, without appreciable effect on her or her child.

Not a single instance of maldevelopment, physical or mental, has occurred in my series.

TOXÆMIAS

We have found with others that in certain toxæmias of pregnancy it was possible to demonstrate focal infections in the sinuses and around the teeth which, when removed, greatly benefited the patient. As a prophylactic measure the X-ray plays an important rôle here as well as in medicine. In cases of hyperemesis treated with the duodenal tube, fluoroscopy will show whether or not the tube is in the duodenum.

MEDICOLEGAL

Operating for tumor and discovering pregnancy requires little comment other than that the physician is usually held liable. Avoidance of unsavory criticism and great expense can be accomplished by resorting to the X-ray when there is the slightest shadow of a doubt.

The medicolegal status of the X-ray in other fields has been established for a long time. In obstetrics its position is still questioned. On the basis of our experimentation, we believe that there is no ground for damages against the physician using it for diagnostic purposes should abortion, fœtal deformities, or failure to have more children, follow its use.

The following shows its importance as a means of protection for the obstetrician:

Within the past year a patient returned to the hospital about 2 months after delivery, claiming inability to walk because of pain in the pelvis. She had been told on arriving home, that she had a fracture of the right pubic bone and a dislocation of the right sacro-iliac joint, brought about by a forceps delivery. Both she and her husband claimed negligence on the part of the hospital and its staff. Immediate roentgenography settled this matter for all time. The pelvis was normal. The hospital and its staff were thereby spared unjust criticism and an incipient malpractice suit was laid low.

This brings us to Lynch's study of pelvic articulations during the latter part of pregnancy and labor. He reported and illustrated separation of the pelvic joints. My study shows this as a rather infrequent occurrence. The plates of Warnekros also show this infrequently, thereby proving that joint separation is neither physiological nor pathological. The former because it does not always show, and the latter because there is no resulting morbidity when it does occur.

CONCLUSIONS

Many in the profession rightly believe that there is more malpractice in obstetrics than in any other branch of medicine. There should be less. The science is rapidly developing. Newer methods are readily being recognized and adopted. There is still much hidden which time and further investigation will disclose. In the meantime there exists this valuable means of conserving maternal and fœtal life (many opponents notwithstanding).

The judicious use of the X-ray in our 250 cases at the Chicago Lying-in Hospital has revealed in the short time that it has been in use a great number of facts which would otherwise have been unrecognized.

Today we can show the student by plates what cannot possibly be demonstrated on the manikin or model, while the obstetrician himself can learn to depend on it as his confrères in medicine and surgery have done; without discredit to his own diagnostic ability but with evidence of his progressiveness.

To Commander Raison of Great Lakes I express my gratitude for his assistance in my pelvimetry experiments.

NOTE.—Since reading this paper, an article relating to X-ray diagnosis of intra-uterine fœtal death has appeared in the June, 1922, number of SURGERY, GYNECOLOGY AND OBSTETRICS.

BIBLIOGRAPHY

ADAIR, F. L. Am. J. Obst., 1918, lxxviii.
ASCHENHEIM, E. Arch. f. Kinderh., Stuttgart, 1920, lxviii, 131–140.
BARTHOLOMEW, R. A. J. Am. M. Ass., 1921, April 2, 912.
CHAMBERLAIN, W. E., and NEWELL, R. R. Am. J. Roentgenol., 1921, viii, 272.
EDLING, L. Muenchen. med. Wchnschr., 1911, lviii, 567.
FORNERO. Ann. di ostet. e ginec., Milan, 1921, xliii, 387.
HESS, J. H. Illinois M. J., 1918, xxxiii, 73–88.
JUDD, A. M. Am. J. Obst., 1915, lxxii, 319.
MULLERHEIM, R. Verhandl. d. deutsch. Gesellsch. f. Gynaek., Leipzig, 1899, viii, 357.
NUERNBERGER, L. Prakt. Ergebn. d. Geburtsh. u. Gynaek., 1920, viii, 163.
O'DONNELL, P. S. J. Am. M. Ass., 1912, lviii, 748.
PANKOW, O. Muenchen. med. Wchnschr., 1920, lxvii, 1016–1032.
WARNEKROS, K. Berl. klin. Wchnschr., 1917, 857.
WEIL, A. E. Paris méd., 1917, xxi, 314; vii, 314.

ADDISON'S DISEASE COMPLICATING PREGNANCY, LABOR, OR THE PUERPERIUM

By GILBERT FITZ-PATRICK, M.D., F.A.C.S., Chicago

ADDISON'S disease is defined by Osler as a disease characterized by muscular and vascular asthenia, irritation of the stomach, and pigmentation of the skin, due either to tuberculosis or atrophy of the adrenals, or to degenerative changes in the chromaffin system generally.

The disease is fairly rare; it occurs less frequently in women than in men, in the proportion of 1 to 9. Only a few reports are found in literature of cases of Addison's disease complicating pregnancy or the puerperium, and in American medical literature I do not find the record of a single case. Altogether in the literature, there are reports of 11 cases, of which short histories are given below. To these I add a case recently observed by myself.

CASES OBSERVED DURING PREGNANCY

Case 1. Reported by Barlow, Lancet, Lond., 1885, i, 251. Patient, age 42, had suffered with the disease for ten months during which time she was pregnant and gave birth to a dead fœtus. The patient had lues of the suprarenals, verified at autopsy.

Case 2. Reported by Jacquet, in Brouardel's Traite de médecine, 1897, iii, 617, refers to a personal case of pregnancy occurring in the course of Addison's disease. Jacquet confines himself to saying that the child was born in good condition at term. During pregnancy pigmentation and other symptoms of Addison's disease were much accentuated, and after labor the points of election of gravidic melanoderma remained intensified. The further course of the disease in this patient is not known.

Case 3. Reported by Miller and Fleming, Brit. M. J., 1900, i, 1014. This was apparently a case of familial Addison's disease. The mother and five children all showed symptoms of the disease. The children in the order of their ages demonstrated all the clinical features in decreasing severity and distinctness. The woman stated that bronzing began after her first pregnancy and became intensified in successive pregnancies. She showed great weakness and prostration, giddiness, diarrhœa, vomiting, pain in the back shooting to the shoulders, palpitation, pain over heart and breathlessness, in addition to the pigmentation and the occurrence of dark moles all over the body.

Cases 4 and 5. Reported by French, Lancet, Lond., 1908, i, 1398. In French's first case the woman was 40 years old and a XI-para. The symptoms of Addison's disease commenced about 6 months before she last became pregnant. There was nothing of note during the early months of pregnancy, but from the fourth month there was weakness of high grade, vomiting, and syncope. As these symptoms showed no signs of abatement the pregnancy was ended at the sixth month. After abortion the pulse rose rapidly, the heart weakened, there were convulsions, and the patient died the second day of the puerperium. Autopsy showed typical caseation of the suprarenal glands.

The second case was very similar according to French but he does not give any details. This patient died also.

Case 6. Reported by Vogt, Verhandl. d. Gesellsch. f. Gynaek., 1913, xv, 115; 249. Woman aged 21 years, primipara. Onset of disease unknown. Course of pregnancy normal. Labor began in thirty-fifth week and it lasted 21 hours, one hour of which was due to the secundines. The fœtus was well developed as an 8-months fœtus, 47 centimeters long and weighing 2,250 grams. The child was living at time of report, being then 14 years old and showed no symptoms of the maternal disease. Like the pregnancy, the birth was without incident, and uterine and abdominal contractions left nothing to be desired. The secundines were discharged normally one hour after birth. No uterine inertia was observed either during or after birth. The puerperium proceeded well for the first 2 days, there was then a sudden thermal elevation the third day, which continued. On the eleventh day vomiting began and could not be controlled, causing collapse and death of the patient on the twelfth day. Autopsy showed both suprarenal capsules caseous, with general anæmia, atrophy of the heart, hyperplasia of the aorta, etc.

Case 7. Reported by Seitz, Inneren Sekretionen und Schwangerschaft, 1913, Barth, p. 191. Woman, age 30, on account of great weakness and augmented pigmentation was suspected of disturbance of the suprarenal glands. In her first pregnancy this woman had shown intense pigmentation of all the body, especially of the genital regions, and this did not quite disappear after the birth. In a new pregnancy, 9 months afterward, the intense pigmentation spread to the arms, trunk, and legs. The bronze color was unaltered a year after the birth.

Case 8. Reported by Guisti, Rassegna d'ostet. e ginec., 1914, xxiii, 465. Primipara; tubercular; 4 months pregnant. The first two months of pregnancy had a normal course. Toward the end of second month mucous and alimentary vomiting, intense gastric crises and general asthenia; face

dark colored; intense pigmentation; asthenia progressively increased. The patient could not stand even for a short time. Blood pressure 100 millimeters. Slight albumin in urine. Diagnosed as pregnancy complicated by Addison's disease. Tubercular reaction positive. Spontaneous abortion at sixth month with dead fœtus weighing 850 grams. Secundines normal. The patient's condition was very grave at the beginning of the abortion, rapidly grew worse, and she died a few days later. No autopsy. The Addison's disease, the author believes, began during pregnancy and he ascribes the suprarenal lesions to tuberculosis.

CASE 9. Reported by Falco, Rassegna d'ostet. e ginec., 1915, xxiii, 434. Age 26 years, XI-para. History of disturbed and scanty menstruation. The cephalæa, gastric crises, lumbar and epigastric pains commonly observed in Addison's disease were frequent in this patient. Abnormal pigmentation. Blood examination: red blood cells, 3,800,000; white blood cells, 6,700; polynuclear neutrophiles, 60 per cent; eosinophiles, 1 per cent; basophiles, absent; small lymphocytes, 32 per cent; large mononuclears, 7 per cent. These findings are similar to those commonly observed in Addison patients. There was constant headache. Pulse 80 to 90; apyretic. Adrenalin was administered without result. The patient gave birth at term to a slightly asphyxiated child. The secundines came 5 minutes later and were normal. No inertia. Uterine and abdominal muscular contractions good. Fœtus weighed 2,820 grams. Lochiæ normal. First days of puerperium, temperature 38.5°, pulse 110-115. The woman nursed her child and left the hospital after 20 days with all the symptoms gone or improved, but 8 days later she died suddenly. There was no autopsy nor could any particulars be obtained of the symptoms before death occurred. The cutireaction for tuberculosis in this case was positive and the author thinks that the Addisonian symptoms were due to a tubercular lesion of the suprarenals. Syphilis may be ruled out.

CASE 10. Reported by Roten, Gynæcol. helvet., Genève, 1915, xv, 113, says that one case of Addison's disease complicating pregnancy was observed at the Basle clinic, but does not give any particulars.

CASES OBSERVED IN THE PUERPERIUM

CASE 11. Reported by Wilkin, Lancet, 1883, i, 498. Age 32; married 3 years; one child which died 3 weeks after birth. Patient has been ailing since birth of child. Examination showed severe epigastric pains, deeply bronzed skin with well-defined chocolate-colored spots about arms and neck, mucous discoloration of lips and cheeks, large dark patch on dorsum of tongue with smaller ones like ink stains. Alteration in her color only became apparent soon after birth of her child. No abdominal pains; great lassitude; and weakness, nausea and vomiting which became aggravated. Died after 3 weeks in bed. No autopsy.

My personal case is as follows:

Mrs. H., white; housekeeper; age 28; I-para; admitted to Maternity Ward, Hahnemann Hospital, Chicago, June 4, 1921. Patient had usual children's diseases; malaria. The family history is negative.

State on admission. At term. Labor began about 11 hours after admission and lasted 4½ hours. The pains were regular and the course and delivery normal. Hæmorrhage was moderate; first degree perineal laceration which was repaired with silk gut. Placenta expelled intact. Uterus contracted and involuted well. Lochiæ normal.

Condition remained good until about 20 hours postpartum. The temperature then rose suddenly from 99° to 102° and continued to rise very irregularly on the days following, reaching 105° on June 10. The condition was thought to be a septic endometritis, secondary to labor.

I saw the patient for the first time June 12. On examining her then the prominent symptoms, besides the high temperature, were: (1) deep bronze color of the exposed skin on arms, chest, and forehead, and especially noticeable over both cheeks; bulging eyes; pupils reacting sluggishly; (2) the speech was only a slow drawl, with very slow grasp of ideas in replying to questions; (3) dullness and sensitiveness over right kidney region; (4) area of dullness over the heart small; (5) abdomen, tympanitic, soft, irregular and sensitive.

The patient showed a systolic pressure of 66; diastolic of 52; pulse pressure 14, pulse 90; temperature 102; respirations 26; respiratory suspensions 25 seconds.

The clinical history showed nausea, continued severe headache, aching feeling over body, severe leg pains, weakness, restlessness, nervousness, giddiness, and inability to sleep; also marked lumbar region sensitiveness and pains.

From the bronze color, the manifest hyposuprarenalism and hypothyroidism, and the clinical history I diagnosed the case as puerperal septic absorption with Addison's disease. Adrenal and thyroid extracts were prescribed.

The temperature variations are shown in accompanying chart.

The administration of thyroid extract was discontinued June 18. Blood cultures were negative for bacteria. Vaginal smears and catheter urine gave some streptococci on culture. Wassermann reaction negative. Blood count showed 17,500 white blood cells; lymphocytes 5 per cent; large mononuclears 1 per cent; neutrophiles 94 per cent.

The study of Addison's disease in pregnancy has an interest first on account of the connection of the suprarenal glands to the sexual apparatus. Disturbances in the glandular secretions cause irregularities or inhibition of menstruation, or sterility. The exact mechanism of the suprarenal secretion on the ovarian

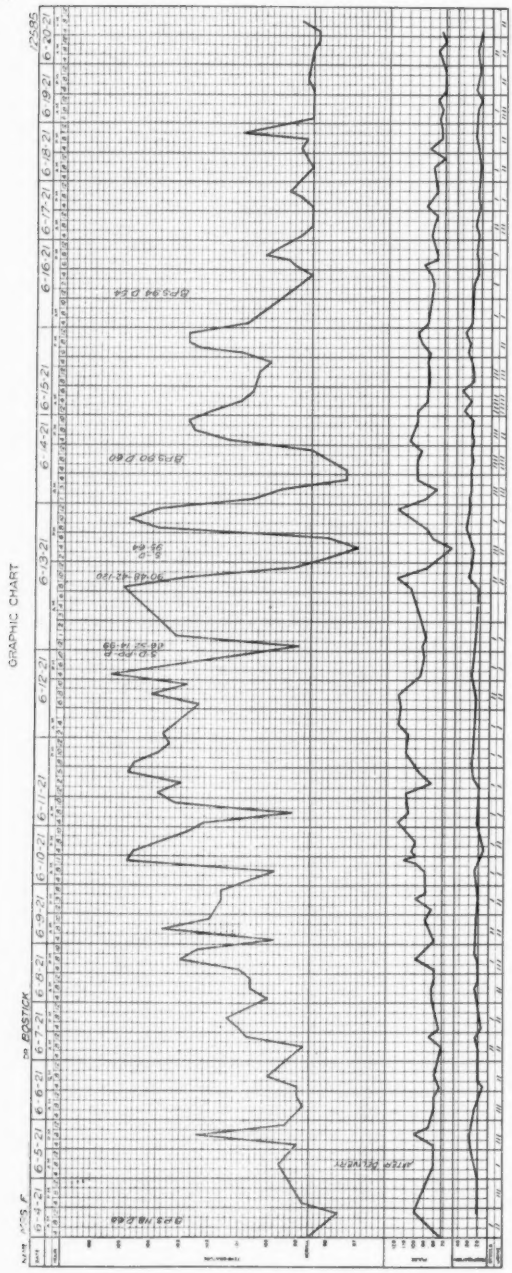

Chart showing temperature variations in author's case.

or other sexual function is not, however, well known. The matter is summarized in Novak's article in Nothnagel's *Specielle Pathologie*, 1912, Supp. vol. I. Physiopathology of the suprarenals has, furthermore, a special interest during pregnancy and the puerperium not only on account of the direct effects of the glandular functioning in these states, but on account of the reciprocal action of the gravid state upon the suprarenal functions. There seems to be little doubt that gravidity demands and effects superfunctioning of the suprarenals as of other glands connected with sexual activity in the cortical zone, though opinions are divided as to whether there is an increased or reduced secretive activity in the medullary zone. Some investigators have found an increased quantity of adrenalin in the blood of the gravid in all periods and attaining its maximum during labor. Others have not been able to substantiate such findings. Whether, in the case of atrophy of the suprarenals, other internal secretory glands act in a compensatory way to supply their particular secretion or to what extent is not known.

We will now consider the reciprocal effects of Addison's disease and pregnancy, labor, and the puerperium.

As regards pregnancy complicated by Addison's disease, cases may be divided into two groups, the first group comprising cases in which the pregnancy occurs in a woman already affected by the disease and the second group those cases in which the Addison's disease appears only in the course of the pregnancy. Guisti's case belongs to the latter type and those of Seitz, Miller and Fleming and the author are doubtful cases. Although the material on which to formulate an opinion is extremely scant, still from what is known and from the theoretical considerations, it would seem safe to assume that pregnancy does not of itself predispose to Addison's disease in a patient whose suprarenals are normal, but that where there is already existing some alteration in the glands pregnancy acts as a stimulus to render a latent Addison's disease manifest; or at least that the effects of faulty functioning of the glands at a time when they are expected to

do even extra work are more manifest in the organism of a pregnant than in a non-pregnant woman. This also applies to cases where pregnancy occurs in the course of Addison's disease, and the intensification of pigmentation and other symptoms during this period, as noted in several cases in the series, supports the view.

The toxins of pregnancy to which are added those produced by the changes in metabolism due to the lack of suprarenal secretion create an unfavorable state of things, debilitating or impeding the normal powers of defense of the organism and consequently accelerating any morbid process, such as a latent Addison's disease.

The influence which pregnancy, labor, and the puerperium exercise on Addison's disease is frankly bad if we judge by the effects in the series of cases quoted. In the 11 cases, 5 of the mothers died before they had reached term. All of these deaths apparently occurred within a short number of days after labor. In the fourth fatal case, the woman went to term but died suddenly 28 days after the birth. The results are evidently grave for the mother and the eventual history of some of the patients in this series is not known.

As regards the child: in Barlow's case the fœtus was dead; French's two cases were induced abortions with dead fœtus; in Vogt's case the child was prematurely born: Guisti's case was a spontaneous abortion with a dead fœtus. If the child lives to term, however, the prognosis is good.

In those who survived the gravid state there were intensifications of the symptoms in the puerperium. In Miller and Fleming's, Jacquet's and Seitz's cases intensification of pigmentation is distinctly mentioned.

From the series of recorded cases it may be deduced that Addison's disease already existing or becoming manifest in the course of pregnancy or of the puerperium has its already bad prognosis aggravated, and its symptomatology intensified.

It is not easy to estimate exactly the effects of Addison's disease on the course of pregnancy, labor, and the puerperium. Experiments on animals have shown that capsulectomy was followed by early abortion in gravid animals. Clinical observations, as in this series of cases, are too few to enable a definite opinion to be formed. While French's, Vogt's, and Guisti's cases ended in abortion, spontaneous or induced, all other cases apparently went to term. An unfavorable influence of Addison's disease on the course of pregnancy need only be anticipated if the disease is very advanced and the patient's general condition poor. In such cases, abortion or premature labor must be the rule, due rather to the general state than to the alteration in the suprarenal glands.

As regards the influence of Addison's disease during labor, in none of the recorded cases was there any noted disturbance during the birth. The uterine and abdominal muscular contractions were noted always as good and sufficient. There was no inertia. If adrenalin plays any important part in the mechanism of labor, the loss of suprarenal adrenalin during labor must have been compensated for in some other way.

The disease seems also to have had very little effect upon the course of the puerperium. The uterus involuted well and the lochiæ were normal in all cases where noted. But it is questionable how far the process of lactation was affected by the disease or how far lactation may have been a factor in the death of the mother, soon after the birth in cases where it occurred. Whatever may be the causes, the puerperal state and lactation seems to have a very damaging effect on the course of Addison's disease. I wish to draw particular attention to one symptom which I observed in my own case and which I do not find mentioned in other case reports. I refer to the drawling, slow speech and decreased mentality. The decreased power of the patient to grasp ideas was very marked.

In my own case the feature that at once struck me when I first saw the patient was the very typical bronze discoloration. The persistent headaches, nausea, and vomiting and the aching all over the body with the feeling of very great weakness, were all in keeping with a diagnosis of Addison's disease, and the subsequent rapid recovery from the symptoms after administration of adrenalin and thyroid extracts confirmed me in my

view. The slow mentality and drawling answers might be considered as due to general asthenia.

Another point which greatly interested me was the abnormal nature of the temperature curve. A study of the temperature charts during the pyretic condition of the patient will show at once that it was not comparable to the ordinary temperature findings in cases of streptococcal or similar puerperal infections but that there was some abnormal factor in this patient's case.

In only 3 of the cases was Addison's disease verified by the anatomical findings on autopsy, viz., in the cases of Barlow, Vogt, and French (first case).

As a practical conclusion, we might seek to know what should be the obstetrical conduct to follow in a case of pregnancy complicated by Addison's disease. As stated already, the disease itself has apparently very little influence on pregnancy, labor, or the puerperium, and when the general state of the patient is good, no interruption need be feared. But if the disease has already affected the general state in an advanced degree, the asthenia may be so great as to bring about a spontaneous abortion or to justify an induced one. The few cases on record do not permit any judgment to be formed as to the necessity for interruption of pregnancy. The maternal prognosis is in any case bad on account of the Addison disease itself which will presumably run its inevitable course. But the fœtal prognosis is good if the child lives to term or nearly so. The child of an Addisonian patient may be born prematurely or at term without showing any of the symptoms which threaten the life of its mother, and in Vogt's case the child has lived 14 years and is well. The obstetrical conduct in ordinary cases should be limited to the medical treatment justified and only to intervene when circumstances absolutely demand it. In the puerperium especially the obstetrician should seek to remove all possible causes which may render it pathological. Lactation ought to be forbidden, as it renders the patient weaker.

The patient has been followed since she left the hospital. The bronze coloration is still evident, especially in the facial region, less marked in the thoracic region, and with one particular spot on the abdomen.

There is epigastric pain easily elicited on pressure but which disappears when the manipulation is continued. In the lumbar region there is an exaggerated tendency to pain over the kidneys especially on deep respiration, and the patient is exquisitely sensitive to pressure over the lower lumbar area. There is some muscular and vascular asthenia.

I may add that the regional pains are not felt even on strong passive exercises of any kind made on the lower limbs. This points to the fact that the pains are dependent upon some deep-seated, rather than on a local, cause.

While any discussion in regard to the etiological factors connected with Addison's disease is outside the scope of this paper, I wish to say that the von Pirquet and complement-fixation tests for tuberculosis have so far given negative results.

REFERENCES

BARLOW. Atrophy of suprarenals with Addison's disease. Lancet, Lond., 1885, i, 251.

JACQUET. In Brouardel's Traite de médecine. Paris, 1897, iii, 617.

MILLER and FLEMING. A family with Addison's disease. Brit. M. J., 1900, i, 1014.

VOGT. Morbus Addisoni und Schwangerschaft. Verhandl. d. deutsch. Gesellsch. f. Gynaek., 1913, xv, 115, 249.

SEITZ. Die Stoerungen der inneren Sekretionen in ihren Beziehungen zu Schwangerschaft. Leipzig: Barth, 1913, p. 191.

GUISTI. Gravidanza complicata da morbo di Addison. Rassegna d'ostet. e ginec., Napoli, 1914, xxiii, 465.

FALCO. Morbo di Addison e gravidanza. Rassegna. d, ostet. e ginec., Napoli, 1915, xxiv, 434.

ROTEN. Kasuistik zur Frage des Morbus Addisonii und Graviditat. Gynæcol. helvet., 1915, xv, 113.

WILKIN. Addison's disease with marked discoloration of the tongue. Lancet, Lond., 1883, i, 498.

FRENCH. Lancet, Lond., 1908, i, 1398.

NOTE.—On June 8, 1922, the family physician, Dr. I. M. Bostick, reports the condition of my patient as follows: Blood pressure systolic, 124; diastolic 84. The bronzing is still well in evidence upon the face and forehead. At times she complains of giddiness with some muscle asthenia and backache. She has steadily gained in weight. She had an attack of influenza in February, 1922, but has apparently recovered without any aggravation of her previous condition.

ADOLESCENT RICKETS

Report of 5 Cases; Osteotomy for Genu Varum and Valgum

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IT is the purpose of this paper to present briefly a discussion of this subject and to show the results obtained in 2 advanced cases: Case 1, a young lady of 23 with severe genu valgum; Case 2, a boy of 16 with genu varum.

Among those who have presented valuable articles should be mentioned Ollier; Goldthwaite, Painter, Osgood, and McCrudden; Poncet and Leriche; Tubby; Robert Jones; Elmslie; Muirhead Little; Lucas; Barber; *et al.* Since the publication of the excellent paper by Horwitz in 1909 there have been comparatively few contributions to the subject. Tubby states that late or recrudescent rickets has been described by Dedeyer and Ollier. Drewitt, in 1880, showed at the Pathological Society in London a case of active rickets in a boy aged 10 years the diagnosis being subsequently verified postmortem. Clutton reported the onset of a rachitic deformity in a girl between 10 and 11 years. He also reported a case in which the deformity came on at 12 years. Cautley had a case in a girl 11 years. She had rickets at 4 years and a recrudescence at 10. In 2 cases of Barlow and Berry the diagnosis was verified postmortem. Jenner referred to a case of rickets coming on in a girl of 19 years. Robert Jones showed a case at The Liverpool Medical Institute. Recrudescent, late, secondary, or protracted rickets is rare. Many deny there is such an entity as rickets originating at puberty and not a recrudescence. There is no *prima facie* reason against it. At this period of life the growth of the long bones has again become very active after a period of comparative quiescence, and, if errors of diet or endocrine disbalance exist, manifestations of rickets may reasonably be expected. Experience proves that cases free from rachitic history, yet showing anything like the generalized epiphyseal enlargements of the active stage of typical rickets, are not often seen. Kirmisson maintains that if the liability to protracted rickets during the whole growing period is accepted, the liability to the onset of the rickets during the same period must be granted. Researches of Mikulicz on the nature of the pathological processes in genu valgum point the same way, and McEwen's views support him. Perhaps the fairest statement which can be made at present is that, although the cessation of the rachitic process may be occasionally delayed to later childhood or adolescence, the primary onset of the disease after infancy is still *sub judice*. It is incorrect to diagnose rickets from the presence of a single local deformity, and the significance of the association of albuminuria and late rachitic manifestations is obscure.

ETIOLOGY

Age. Adolescent rickets, as its name implies, is rickets occurring during adolescence. There is considerable discussion of the question as to whether it is a definite entity, that is a condition *de novo*, or a recrudescence of an infantile condition that has remained dormant during a period of comparative osteogenetic inactivity. In the 5 cases herein reported, the age of onset was 14, 14, 16, 10, and 18 years respectively.

Sex. In the experience of the writer, females are more prone to this affection than males. Females are more apt to develop scoliosis; males, deformities of the extremities.

Heredity. There is no definite evidence upon which to base an hereditary etiology.

Color. In contrast with infantile rickets, none of the cases in this report occurred in a negro.

Nationality. It seems noteworthy that none of the present series of cases occurred in children of Italian, or Syrian parents, among which peoples so large a proportion of infantile rickets occurs.

Infection. In only 1 of the cases is there any mention of infection. That is, in Case 1 the knock knees were made worse by an attack of influenza. It appears to have had only a casual relationship.

Trauma is evidently of no great importance.

Metabolism. In the writer's opinion it is entirely a question of bone metabolism. However, the subject of metabolism is very closely allied with two important factors, viz., endocrinology and vitamines.

Endocrinology. It seems that there occur, at the time of sexual development, certain as yet somewhat indefinite changes in all the endocrine glands through the interrelationship and inter-activity of which there is produced a disturbance of osteogenesis resulting in deformity. The explanation of the exact nature of these changes is speculative. For example let us consider the case of the female. Until the age of 14 years all her endocrine glands are functioning normally and a "balance of power and effect" obtains. At the age of 14 her ovaries take on their normal function, develop, and add their internal secretion; and this disturbs the "balance." It is possible that excess of ovarian secretion stimulates the thyroid which in turn activates the parathyroids, thereby causing an increased output of calcium which renders the long bones softer and they bend.

Involution of the thymus coincides normally with adolescence, and disturbed thymus balance might cause bone and muscle changes.

PATHOLOGY

The pathology of adolescent rickets concerns itself practically entirely with the gross changes occurring locally in the parts affected, viz.: knees, hips, spine, feet. In case of renal dwarfism, the nephritis is important. As no tissue was removed from either operative case, no microscopic description can be given.

In genu valgum the inner condyle of the femur is markedly enlarged and the joint surface slants downward and medialward. In genu varum the opposite condition is found.

In genu valgum the muscles and capsule on the inner border are markedly stretched and weakened. The internal lateral ligament and crucial ligament are also relaxed. The corresponding tissues of the outer border of the joint are shortened. In genu varum the stretching and weakening are on the outer border of the knee. According to Keith's law of ligaments, no ligament is ever used for the continuous support of any joint or part.

If the muscles on the inner side of the knee give way, the strain falls on the internal lateral ligament, the ligament lengthens, as every unsupported ligament must do when subjected to continuous strain: so do the parts of the femur and tibia to which the ligament is attached. A small, then a larger, gap appears between the articular surfaces of the internal femoral condyle and inner tuberosity of the tibia. The condition of knock-knee is thus produced, because the abnormal strain on both the internal and external femoral condyles alters the rate of growth in the outer and inner portions of the epiphyseal lines. If, on the other hand, the outer muscular supports give way, a bow-leg is produced.

The primary cause of static deformities of the knee lies in a defect or change in the osseous system, but the structures which are immediately responsible for the deformity are the muscles around the joint. In a rachitic child the muscles are flabby and have lost their reflex tone, the ligaments of the knee joint have thrust upon them a static muscular function with the result that deformities occur. If these deformities are to be prevented, the lower extremities must be relieved of weight bearing until the bones have regained their normal health and rigidity. The reflex tone of a muscle depends upon its state of tension, which state depends upon the absolute rigidity of the bones on which they act. As soon as the normal bone rigidity has returned, the muscles resume their normal static, reflex function.

SYMPTOMS

Deformity. The symptom which brings the patient to the hospital is deformity: usually genu valgum or varum.

Pain. When the deformity has existed for some time, there may develop pain.

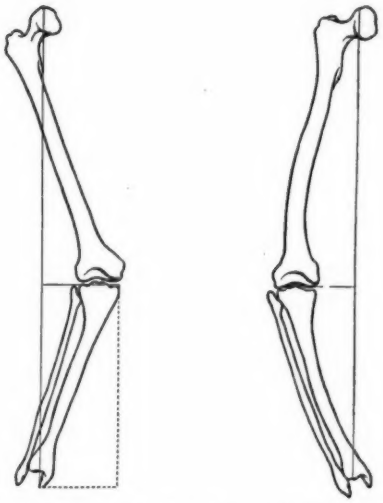

Fig. 1. Schematic drawing of genu valgum and of genu varum. (After McEwen.)

Weakness is due to the mechanical disturbance caused by the deformity.

Decrease in height is due to the deformity.

The cosmetic effect of the deformity especially in the girl is the inability to wear short skirts.

The X-ray clearly defines the deformity but the gross bony changes are not apparent.

DIAGNOSIS

The diagnosis is easy when a good history is obtainable and a physical examination is made, and the X-ray corroborates the diagnosis.

Osteomalacia occurs in older people. It is common in females and very rarely found in males. (See report of Kanavel, Elliott, and Nadler.) It is a generalized condition. There is marked absorption of lime salts as shown by X-ray.

Paget's disease (osteitis deformans) occurs in older people, usually past 40, and more commonly in males. It is usually a generalized condition, i.e., the following syndrome is present: bowing of a leg, increased size of the skull, cervicodorsal kyphosis, diamond-shaped abdomen, simian type, and areas of absorption in the long bones as shown by the X-ray. (There is a monosteitic type.)

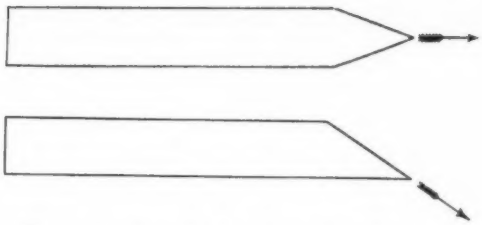

Fig. 2. Drawing, showing shape of cutting edges of chisel (below) and osteotome (above).

PROGNOSIS

The prognosis as to life is good. The writer has not found in the literature any statement to the effect that adolescent rickets shortens life. Barber warns against osteotomy in renal dwarfism because 1 case died 10 days after osteotomy. He believes uræmia is inevitable: in 7 fatal cases the ages were 11, 14, 14, 15, 15, 16, and 16. His oldest case alive is 20 years. Blood-urea determination and kidney function tests should be of value in making a prognosis in these cases.

As to correction of the deformity, the prognosis is very good.

TREATMENT

The writer's experience advises against the use of the osteoclast and recommends osteotomy.

Technique. The field of operation is carefully prepared on the preceding day. At the time of operation, another preparation is done and over the point selected a small incision is made and a chisel (not an osteotome, see Fig. 2) is inserted. This is worked back and forth parallel with the long axis of the bone until it pierces the periosteum. Then, with the chisel pressed very firmly against the cortex, the cutting edge is turned through an angle of 90 degrees until it lies transverse to the axis of the bone. A piece of gauze is wrapped around the chisel and the instrument held very firmly against the bone. By means of a mallet or a well-balanced potato masher the bone is chiselled about three-quarters the way through. The remainder of the fracture is accomplished manually. A green stick fracture is highly desirable. If a complete fracture is made, it is important

Fig. 3. Supracondylar osteotomy.

that the fragments be held so as to prevent prodding of the muscles, which is always a source of much discomfort. The wound is closed with catgut subcutaneously and either catgut, silkworm, or waxed silk cutaneously. Now the stockinette (which had been rolled up near the groin before the operation was started) is pulled down and the limb encased in a layer of sheet wadding and roller bandage. Felt pads protect the iliac crests, knees, malleoli, heels, and points of fracture, and a plaster-of-Paris cast is applied. As the cast sets, slight overcorrection of the deformity is obtained. If the fractures are above the knees a double spica is necessary; if below, the cast should extend from the perineum to the toes. The legs should be elevated in bed.

As part of routine these patients are given (1) phosphorus and cod-liver oil[1]—phosphori gr. 1/6, ol. morrhuæ ʒii—1 teaspoonful 3 times daily before meals; (2) a high vitamine diet containing as much of the following as possible: cream and butter, spinach, tomatoes, cabbage, onions, beets, beans, peas, turnips, carrots, orange juice, lemon juice, crisp bacon, fresh fruits. In this diet all 3 groups of vitamines are included: fat solubles (a) and (b), and water solubles (c). (3) As much sunlight and fresh air as possible.

Castration is usually not indicated. In extreme, acute case there might be a possible indication. This procedure has been recommended because of its value in some cases of osteomalacia.

[1] Phosphorized cod-liver oil is excellent because the dosage is more reliable.

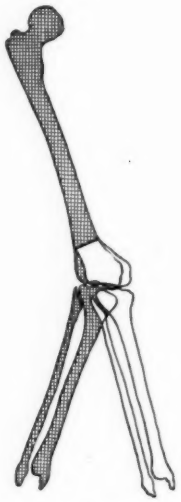

Fig. 4. Osteotomy of femur. (After Ollier.)

It seems rational to administer some of the endocrine glands in selected cases, especially thyroid and hypophysis.

Wolff's law. This law applies to adolescent rickets. Quoting from Freiberg's review of Wolff's article: "Every change in the form and function of the bones or of their function alone is followed by certain definite changes in their internal architecture, and equally definite secondary alterations of their external conformation in accordance with mathematical laws." In a few words it means, "function determines form." Therefore, after correction of the deformity, as recommended above, the internal and external forms of the bones become more normal.

CASE 1. F. McD., white girl, age 23, born in Chicago, of Irish parentage, referred by Dr. James H. Hutton. Admitted to St. Luke's Hospital, February 20, 1921, complaining of knock-knees, pain in knees; difficulty in walking.

Patient has been told that she had rickets at the age of 3 years. She had no trouble with her legs until she was 12, when she began to menstruate. About this time she was compelled to lift heavy baskets of clothes and do heavy work. Then she noticed stiffness and pain in knees. When about 14 years she noticed she was getting knock-kneed. During this time she had stiffness and pain in knees and experienced difficulty in walking, at times finding it necessary to hold on to things as she

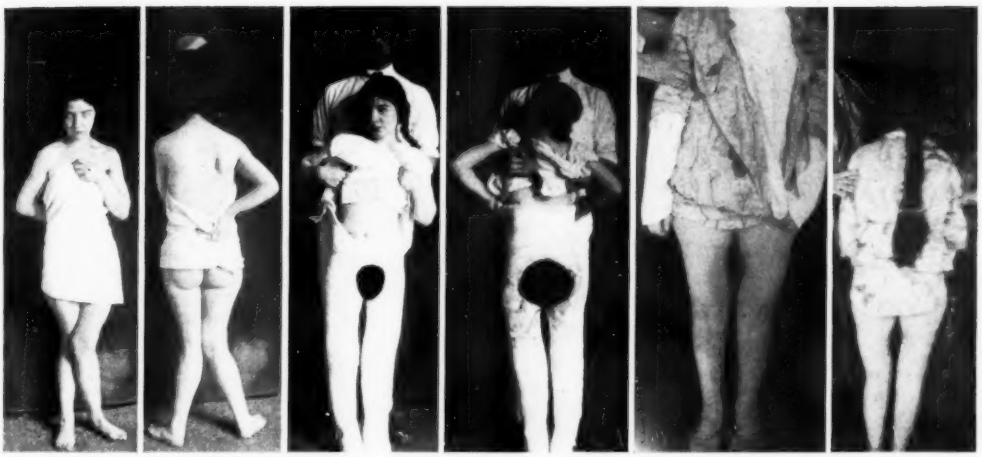

Fig. 5. Fig. 6. Fig. 7. Fig. 8. Fig. 9. Fig. 10.

Fig. 5. Case 1. February 20, 1921, before operation.
Fig. 6. Rear view before operation.
Fig. 7. Case 1. April 15, 1921, 6 weeks after operation.
Fig. 8. Case 1. Six weeks after operation.
Fig. 9. Case 1. Five months after operation.
Fig. 10. Case 1. Five months after operation.

walked. From 14 until 21 years the stiffness and pain gradually disappeared and the knock-knees remained stationary. Two years ago she had a severe attack of influenza, since which time the pain has returned but there is no stiffness. She has become more knock-kneed, and walking is increasingly more difficult. The knees become so weak while walking that she is forced to rest.

Patient has had measles and rickets, and influenza 2 years ago. She has worn braces for her legs. Venereal infection is denied. Menses began at 12 and have been of the regular, 28-day type, duration 3 to 4 days. Dysmenorrhœa inconstant.

Patient knows very little of her family, which accounts for inability to give early history.

Patient uses tea and coffee but no alcohol. She frequently had diarrhœa in childhood but is troubled with constipation now. She has not been troubled with nocturia, with frequency, or with irritation. She weighed 117 but now weighs 103¾ pounds. She is 4 feet 9 inches tall.

Examination shows the upper extremities normal. There is a bilateral genu valgum of the lower extremities, much worse on the right side. When the internal femoral condyles are approximated, the malleoli are 11 inches apart. There is also bilateral hallux valgus of moderate degree.

On February 21, 1921, the urine showed albumin, hyaline casts, red cells, and leucocytes. On February 22, 1921, a supracondylar osteotomy was done, and the femora placed in a double spica cast. On March 19, 1921, the patient rested on a stretcher in the yard. On April 28, 1921, she was discharged with casts on, to return in 1 month. She is about 2 inches taller than before the operation.

A roentgenogram on March 22, 1921, made by Dr. E. L. Jenkinson, showed an oblique fracture through lower third of left femur. The upper fragment was displaced outward and slightly backward. There was a transverse fracture of the right femur in lower third. The upper fragment was displaced slightly outward. Lateral position was very good.

Basal metabolism test showed +1.3.

Case 2. Joe G., white boy, age 16, born in Chicago, of Polish parentage. Admitted to Cook County Hospital, October 28, 1920, complaining of bow-legs.

Patient states that he had "bow-legs" when a young child but that he outgrew them. A photograph of him at 13 years shows straight legs. At about 14 years of age when adolescence began he noticed that his legs were bowing. (At this time he was doing heavy lifting as a grocery boy.) The condition has gradually increased ever since.

The past medical and surgical history is negative. The father, mother, brother, and sister are living and well; 2 sisters and 1 brother dead, cause unknown.

Patient attended public school until he reached seventh grade, at 14 years. He is employed in a factory and his work requires that he keep sitting position. He sleeps and eats well and has been increasing steadily in weight during past 2 years.

Physical examination shows a young white male of about 16 years, whose legs are markedly bowed.

There is a marked bowing of both legs, the right much worse than the left. The upper tibiæ are involved chiefly but the femora are also affected. While standing with heels and toes touching, the internal condyle of the right femur is 3½ inches

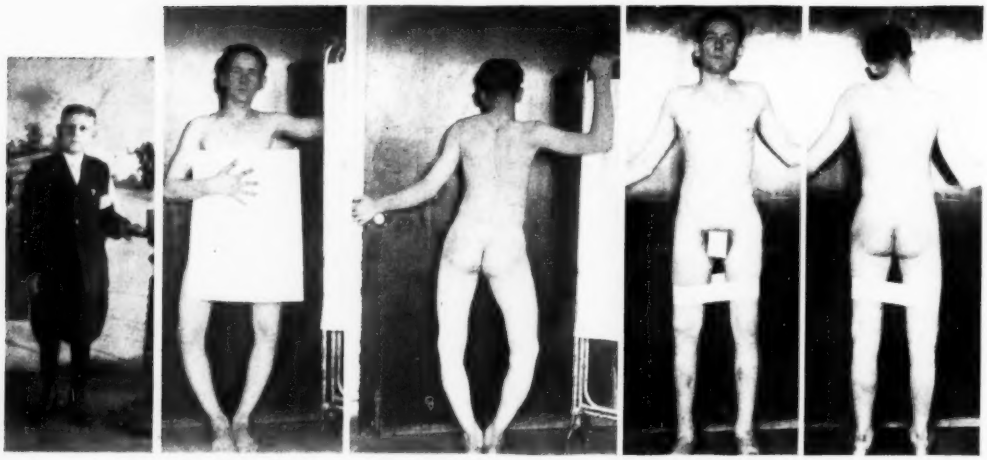

Fig. 11. Fig. 12. Fig. 13. Fig. 14. Fig. 15.

Fig. 11. Case 2. Legs straight at age of 13.
Fig. 12. Case 2. Showing large hand.
Fig. 13. Case 2. Before operation.
Fig. 14. Case 2. Standing alone, 6 weeks after operation.
Fig. 15. Same as Figure 14.

from the midline of the body, and the left is 2½ inches from that line. There is a 10-degree bilateral genu recurvatum. Decidedly pronated feet.

Movements are normal.

General examination reveals no pathology.

Diagnosis: Genu varum, genu recurvatum, pes planus.

X-ray examination shows:

1. No X-ray evidences of bone or joint changes in the several regions examined. The sella turcica seems very much flattened and shallow. No material increase or decrease is seen (Blaine).

2. No marked evidence of remaining rickets except the curvature of the long bones. The sagittal suture in anteroposterior projection does not seem to be firmly united. Longitudinal arches of feet are depressed (Blaine).

3. No evidence of the cause of the bow-legs. Bone densities are approximately normal.

4. Practically all the epiphyseal lines closed and the individual bones of the pelvis and lower extremities more than usually large. A certain coarseness of appearance is noted. No localized bone changes are observed (Blaine).

5. After operation, apposition of fragments good. There is an equal degree of slight internal angulation at the point of each fracture (Matthews).

July 12, 1921. Metabolism test normal.

July 22, 1921. Operation. Open osteotomy of tibiæ. Plaster-of-Paris casts. Slight overcorrection.

CASE 3. J. McC., white girl, age 22, born in Chicago, of Irish parentage. Seen at South Side Free Dispensary, Northwestern University Medical School. Until she was 16 years old her legs were normal. At that age she began to menstruate and knees began to bow. Comes for treatment of coccygodynia.

CASE 4. R. F., white girl, age 17, born in Indiana, of Russian parentage. Entered St. Luke's Hospital on the service of Dr. John L. Porter (for whose permission to report this and the next case, I am very thankful). Genu varum was well marked. It was first noticed at age of 10 years. Her father is bow-legged also. Menses began at 13. Operation: Osteotomy of tibiæ and forcible correction.

CASE 5. A. E. K., white boy, age 19, born in Chicago. Private patient of Dr. John L. Porter. Genu varum, left, well marked. General health and nutrition excellent. About 1 year previous to date of entrance he noticed that his left leg was "out of line." He had no explanation of the cause. At the onset of the trouble, there was some pain but this gradually disappeared until the present time when he has no complaint except the outward bowing of the left lower leg. He grew very rapidly during the past few years and simultaneously engaged strenuously in athletics in high school. A tentative diagnosis of either adolescent rickets or beginning monosteitic Paget's disease was made. X-rays made by Dr. Hollis E. Potter revealed elongation of the inner condyle of the left femur, but no other bone pathology. No operation indicated.

CONCLUSIONS

It is a very interesting fact that these deformities either make their appearance, or, if present, grow markedly worse, coin-

cident with adolescence. During this period increased demands are made on the organism, and if at that time the general metabolism and especially bone metabolism is disturbed, deformities are prone to occur. It is probable that a combination of bone softening, plus heavy lifting or carrying, causes some cases. Others are due to relaxation of muscles and ligaments, and in this type infections may be a causative factor.

Certain definite factors occurring at a certain definite "cycleogical" time. These factors are:

1. The growing period for that individual;
2. Athletic overindulgence, as seen in the high-school boy;
3. Physical overwork, such as carrying heavy loads as in the case of the grocery boy, or the young girl doing domestic work which is too strenuous for her;
4. General food deficiency;
5. Particular food deficiency;
6. Stimulated endocrine activity;
7. Suppressed endocrine activity;
8. Disturbed endocrine activity, i.e., abnormal chemical secretions.

It is the writer's belief that the whole question is one of bone metabolism and especially calcium metabolism due to the above-mentioned factors, and the immediate cause of deformity is a disproportion between the carrying-power of the bones and the load they are forced to carry.

The writer believes that corrective measures should be instituted and that operation is not attended by unusual risk.

The writer desires to thank those whose names are mentioned in this paper and especially Dr. John L. Porter for the privilege of reporting Cases 4 and 5 and Dr. J. H. Hutton, Case 1.

REFERENCES

BARBER. Quart. J. Med., 1921, xiv, 205; abstracted, J. Am. M. Ass., 1861, lxxvi, No. 26.
BLANCHARD. Am. J. Surg., 1921, Sept.
CAUTLEY. Brit. M. J., 1896, Jan. 4.
CLUTTON. St. Thomas Hosp. Reports, 1884, xiv, 103.
DREWITT. Tr. Lond. Path. Soc., 1881, xxxii.
ELMSLIE. St. Bartholomew's Hosp. Reports, 1906, xlii.
ENGELBACH and TIERNEY. Tice's Practice of Medicine, vol. viii.
ESSER. Muenchen. med. Wchnschr., 1907, xvii; Boston M. & S. J., 1907, June 20.
FEHLING. Arch. f. Gynack.
FINDLAY. Brit. M. J., 1908, July 4.
FREIBERG. Ann. Surg., 1897, July; Am. J. M. Sc., 1902, Dec.
GOLDTHWAITE, PAINTER, OSGOOD, and MCCRUDDEN. Am. J. Physiol., xiv, No. 15.
HENZE. Abstracted, J. Am. M. Ass., 1921, lxxvii, 1378; Ztschr. f. klin. Med., Berl., 1921, xci, 120.
HORWITZ. Am. J. Orthop. Surg., 1909, vii, 199.
HUTCHISON. Brit. J. Child. Dis., ii, 222.
JENNER. Med. Times & Gaz., 1860, i.
JONES, R. Brit. M. J., 1896, Feb. 7, p. 341.
J. Am. M. Ass., 1921, Aug. 13, p. 571.
KAREWSKI. Die chirurgischen Krankheiten des Kinderalters. Stuttgart, 1894.
KEATINGS. Cyclopedia of Diseases of Children, vol. ii, 225.
KEETLEY. Illus. M. News, 1888, Sept.; Ann. Surg., ii, 308.
KEITH. Menders of the Maimed. London: 1919, p. 166.
KIRMISSON. Les Difformités acquises, p. 216.
LEWIN. J. Bone & Joint Surg., 1922, iv, 45.
MARPURGO. Ztschr. f. orthop. Chir., xviii, 52.
MARSDEN. Brit. J. Child. Dis., ii, 565.
MARSHALL. Proc. Roy. Soc. Med., 1920, xiii, Sect. Surg., 229.
MCEWEN. Osteotomy Tr. Demons., 1882; Osteotomy. London: J. & A. Churchill, 1880.
NICHOLS. Keen's Surgery, vol. i.
OLLIER. Thésis de doct., Lyons, 1895.
PALM. Practitioner, Lond., 1890, xlv, 275.
PATTERSON. Proc. Royal Soc. Med., 1911, iv, Child. Sect.; 1912, v; 1920, viii; 1920, ix.
PHEMISTER. J. Am. M. Ass., 1918, lxx, 1737.
POMMER. Osteomalacia u. Rachitis. Leipzig: 1885.
PONCET and LERICHE. Bull. de l'Acad. méd., Par., 1907, Oct.; J. Am. M. Ass., 1907, Nov. 23, p. 1810.
RANSFORD. Brit. M. J., 1887, i, 1213.
ROOS. Ztschr. f. klin. Med., 1903, xlviii, Nos. 1 and 2.
ROTTER and LAUENSTEIN. Arch. f. klin. Chir., 1890, xl.
SAUER. Abstracted, J. Am. M. Ass., 1921, lxxvii, 239.
TOSHIRO. Tr. Med. Soc., Tokyo, 1906, Oct. 19.
TUBBY. Deformities, vol. ii, 400.
WHITMAN. Orthopædic Surgery, p. 493.

CHRONIC BONE ABSCESS

Its Treatment by Simple Evacuation through a Drill Hole

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From the Surgical Services of the Mt. Sinai and Broad Street Hospitals

IN an earlier communication on "Chronic Medullary Abscess of the Long Bones" (1) I called attention to the facts that these abscesses, usually from residual foci of an antecedent osteomyelitis, often become sterile, and that such abscesses can be promptly cured by the minor operation of simply evacuating them through a small opening in the bone.

In that communication I said: "The medullary abscess, like acute suppuration of the bone marrow, cannot be recognized as such, roentgenographically. The roentgen-ray appearance is that of an osteoperiosteitis or of a bone that has been the seat of an osteomyelitis." The statement is not wholly correct. When, as sometimes happens, a considerable length of the medullary cavity is occupied by pus, there is nothing in its roentgenographic picture to show that its content is purulent. On the other hand, when the medullary cavity is invaded by osseous growth in such a manner as to surround an "island" of pus, such a central abscess will appear in the roentgenographic positive as a light area, by contrast with the shadow of the surrounding bone—unless, indeed, as may also happen, the latter is so thick and so dense that it obscures the picture of the cavity within it. Cortical abscesses, of course, usually show clearly in X-ray pictures.

The technique of my procedure, as developed, is as follows: Under nitrous oxide narcosis, an incision about 2 inches long is made through the skin and (except, for example, over the inner surface of the tibia) the underlying muscle is split, or retracted, to expose the periosteum. Through this and into the bone, at a point determined by roentgenogram or by the level of maximum tenderness, a drill about one-fourth inch in diameter is driven deep enough to permit the escape of pus. If, perchance, the situation of a small abscess has not been estimated sufficiently accurately for it to be thus tapped by the first opening, other drill holes can be made with little damage to the bone, or the surgeon may defer the completion of the operation to determine roentgenographically the relative levels of the abscess and his first drill hole. With the escape of pus the drill is removed and *nothing else is introduced into the bone*—no probe, curette, or gauze packing! A culture is made from the pus to determine what, if any, living organism it contains. A smear is also stained and examined at once. The purpose of the latter is that if, instead of few or no bacteria, many are found, the surgeon may, if he thinks it desirable, enlarge the bone opening or otherwise modify the procedure. A small drain of folded rubber dam is laid in the soft parts down to, but *not into*, the opening in the bone, and the wound is otherwise closed by sutures. A simple gauze dressing completes the operation.

The drain is to provide for any further escape of pus from the bone, and to act as a safety-valve in case of possible suppuration in the bone cavity or the soft parts caused by dormant organisms awakened to activity. It is removed, usually without replacement, in 3 or 4 days, i.e., as soon as discharge has practically ceased. The small opening in the soft parts is then allowed to close and, ordinarily, healing of the entire wound is complete in 10 days or less, but few of which, usually, are spent in bed. Contrast this with the prolonged disability and invalidism, not to speak of the scarring and deformity, of wide-open osteotomy and slow "healing from the bottom," with its inherent risks of sequestrum formation and secondary operations!

The pain of a chronic afebrile bone abscess which, with tenderness, is the sole subjective symptom, is relieved by evacuation of the pus through a drill hole, immediately, completely, and—as far as my observation of several

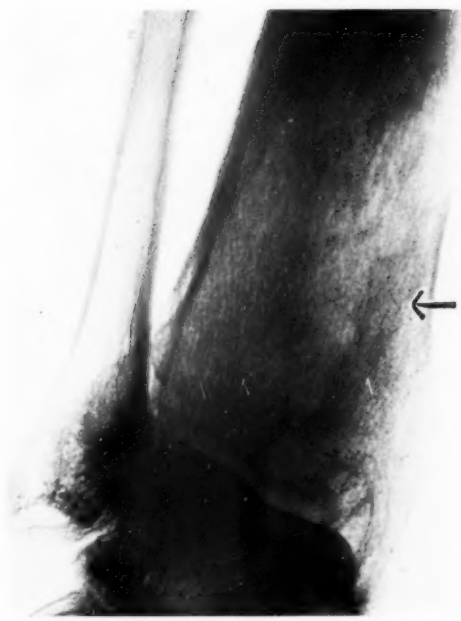


Fig. 1. Dr. A. B. Abscess in the lower end of the tibia twenty years after acute osteomyelitis. Note that there is very little sclerosis of surrounding bone.

Fig. 2. Dr. A. B. Eighteen months after evacuation of the abscess through a small drill hole. Note that the cavity is not entirely obliterated.

years warrants me in saying it—permanently. Failure thus to relieve the pain suggests the presence of a second abscess; such was the condition in a case, treated by my method, reported to me by a colleague.

Failure of prompt healing of the soft parts will, of course, arise if there be infection either from the operation itself or from bacteria in the evacuated pus. I have had one such experience, but the infection, a mild one, subsided under wet dressings without further operation.

Since the publication of my previous article reports of recent operations for chronic bone abscess have been made by Bevan, by McWilliams, and by Bancroft.

Bevan (2) presented two adult cases of small deep, chronic bone abscess, both recognizable roentgenographically. One of these, in the tibia, followed an osteomyelitis 33 years before; the other, in the femur, appears to have been quite like Case I in my first paper and, like it, without antecedent history of bone or other infection. In both cases Bevan made a 5-inch incision, freely "gouged out" the bone, gouged and curetted the abscess cavity, flushed the wound with hydrogen peroxide and then with alcohol, and at once sewed the soft parts completely. In both cases these healed *per primam*. Both patients were kept in bed 10 days but it "requires another 5 or 6 weeks for the repair in the bone to be completed." The procedure is one Bevan had been employing for a long time.

McWilliams (3) reports a chronic central abscess of the tibia, with pain for 2 years, probably resulting from a stab wound of the bone a year earlier. Through a six-inch incision he chiselled a furrow 3 or 4 inches long, in densely sclerosed bone, down to the medullary cavity, from which he evacuated an abscess of the diameter of a quill and 3 inches in length. The upper part of the wound was closed by sutures, the lower part was left open for three Carrel tubes. On the fourth day of dakinization "no organisms were found in

smears." The wound was then entirely closed by secondary suture. It healed promptly and the patient was discharged, cured, on the twelfth day.

As compared with the generally practiced tedious treatment by open osteotomy and repeated gauze packing, both Bevan's and McWilliams' methods of treating chronic bone abscess are distinctly steps in advance. But that they both secured early healing—one after primary, the other after secondary, suture—confirms my faith in the rationale of the still simpler method that I have advocated. In the absence of any statement to the contrary, it is presumable that both of Bevan's patients were afebrile—as is so commonly the case in this affection. He mentions no smears from the pus and no cultures. It seems to me quite likely that he secured primary union, not because of his vigorous mechanical attack upon the bone and his chemical antiseptics, but because the abscesses were sterile. McWilliams' patient was also, presumably, afebrile. He refers to no smear examination of the pus and "the cultures taken at the time of the original operation were contaminated, so the causative organism is unknown." In his case, too, the abscess may well have been sterile or nearly so.

Bancroft (4) reports a small, chronic, medullary abscess in the upper third of the tibia, which, like McWilliams, he treated by dakinization—without, however, the important detail of secondary suture. In his case there had been a localized thickening of the bone for 17 years. The pain present at the outset of the disease soon ceased, but it had recurred in recent years, was intermittent, not severe, and not associated with tenderness. Bancroft chiselled through dense bone, evacuated thick pus from a cavity about 1 centimeter in diameter, curetted this cavity, trimmed off the overhanging bone, and sutured across the space the periosteum and the skin, leaving an opening in these tissues large enough for four Carrel tubes. How long these tubes were maintained in the wound is not stated. The patient left the hospital on the sixteenth day, but the discharge continued for another 3 weeks. In effect, Bancroft's treatment was by free osteotomy and continued drainage through a partially sutured wound. The pus in his case contained staphylococcus aureus in pure culture. Whether or not his patient had any febrile elevations he, too, does not state.

I am quite prepared to believe that chronic bone abscesses unassociated, over a limited period of observation, with any elevation of temperature, are not always sterile. But it seems safe to assume that their contained organisms are attenuated. We can hardly depend upon killing contaminating bacteria in the freshly cut surfaces of bone, muscle, fascia, and skin by a single treatment with hydrogen peroxide and alcohol.

In McWilliams' case there was no tenderness, and no abscess was shown by the roentgenogram. It did show, however, a fusiform thickening of the bone 3 inches long. Since the abscess within it was also 3 inches long, it could have been tapped at any level in that area—a procedure which seems to me preferable, in these cases, to the osteotomies performed by McWilliams and by Bevan, with their greater risks of later trouble.

Simple evacuation through a drill hole is not at all suitable, of course, for those cases of non-discharging bone suppuration with high fever. In these cases we are dealing, in my experience, no longer with a "chronic bone abscess," if at all, but with a (recrudescent) acute osteomyelitis. At any rate, the process is a virulent one and demands vigorous surgical attack. Whether the method is suitable for chronic typhoid abscess I do not know.

How far it is applicable to those cases of chronic bone abscess in which clinical signs—daily mild elevation of temperature, slowly increasing pain and tenderness—indicate that the pus is not sterile but that the virulence of the organisms is slight, my experience is too limited to say. It has been sufficient, however, to make me offer the suggestion that in these cases simple evacuation through a small opening is well worth a trial, provided the patient is under close observation. It must be remembered that, although the bone opening is relatively small, drainage is provided. The following case is illustrative:

Dr. A. B., dentist, age 32, referred to me August 4, 1920, by Dr. Philip Hirtenstein, was operated upon at the age of 11 for acute osteomyelitis of the left

tibia. The disease left the bone irregularly thickened, and fixed by osseous ankylosis to the femur, at which level there was a small cavity discharging from time to time through two sinuses.

For a fortnight Dr. B. had noted gradually increasing pain and "heat" in the region of the lower end of the left tibia. There was localized tenderness on pressure over the inner aspect of the expanded lower end of the tibia, suggesting the diagnosis of chronic bone abscess. The noon temperature that day was 99.5°.

On August 13 he arranged to enter Mt. Sinai Hospital. During August 13 to 15 it was noted that the evening (rectal) temperature was 100.5°–101°, that the pain had somewhat increased and that there was, as before, a point of great tenderness when the inner surface of the tibia was pressed upon 2 inches above the tip of the malleolus. Roentgenograms showed the familiar picture of a long bone that has been the seat of an extensive osteomyelitis. Much of the medullary cavity, especially in the lower half, was invaded by bone, but in the lower end, at the level of the area of tenderness, was seen, in the center, a somewhat irregular cavity about the size of a Jordan almond. This confirmed the diagnosis of abscess. It was interesting to note that the bone adjacent to the cavity showed only *very slightly* increased density posteriorly; elsewhere the bone circumferential to the abscess and, indeed, in a considerable area beyond, was of normal density and its markings appeared very clearly (Fig. 1.). There was slight periosteal thickening over the shaft of the bone posteriorly, probably old, for it is still present (Fig. 2).

August 16, operation. An incision about one-and-a-half inches long was made over the inner aspect of the lower end of the tibia, and the tendons retracted. At the point determined by roentgenogram and by maximum tenderness, a one-eighth inch drill was driven to the center of the bone. Thick pus escaped. Smears showed that it contained a few cocci; and cultures developed a pure growth of staphylococcus aureus. Tincture of iodine was injected into the cavity. The middle of the skin wound was left unsutured; since the bone is here subcutaneous, no drain was needed. Wet dressing.

For 2 days succeeding the operation the evening temperature was 103°, the lower leg was moderately swollen (there was a distressing iodine skin reaction), and there was slight purulent discharge from the wound. During the next 2 days the temperature fell gradually to normal, where it remained, the swelling disappeared, and the suppuration ceased.

On September 9 the patient left the hospital, walking. The wound was then small and granulating normally. It contracted to a granulating, practically dry sinus, leading into the bone. This remained until, about 9 weeks after the operation, there escaped a thin, small, circular sequestrum, which had formed about the drill opening. The sinus then promptly closed and has remained healed. There has been no recurrence of symptoms. A roentgenogram made 18 months after operation shows that the drill hole is closed by bone, but that the abscess cavity, although reduced in size, is not obliterated. The periosteal shadow is unchanged (Fig. 2).

McWilliams makes no mention of my method of treating chronic bone abscess, and refers to my earlier communication, which dealt only with the medullary type, as an "article on Brodie's abscess." On the basis of Brodie's description he is quite justified in doing so. Several generations of medical students have been taught, however, to think of Brodie's abscess as a circumscribed collection of pus in the bone cortex, usually near the epiphysis. To apply the term without distinction to all chronic non-discharging pus pockets, in whatever part of the bone, may serve only to obscure the student's conception of the disease. It seems to me better to speak of "cortical abscess," which is pictured in all the textbooks (as Brodie's abscess) and "medullary" or "central abscess," to which but few textbooks even refer. Indeed, in the latest, very recent edition of his work on *Surgical Pathology*, Sir Anthony Bowlby again makes the statement: "Chronic abscess is never met with in the shafts, but always in the cancellous tissue of the epiphysial ends." We should eliminate from our future textbooks the statement that Brodie's abscess is usually tuberculous. It is of a piece with the lingering notion that fistula-in-ano is commonly tuberculous.

Concerning the pathogenesis and pathology of chronic bone abscess there is much that is debatable. The pain is, presumably, due to the pressure of the pus, since it disappears as soon as this is drained. If, however, the abscess was present before it caused pain, what produces increased tension at a time when no organisms are recoverable from the pus? It seems to me more likely that the pain begins when pus is formed in a cavity containing dormant bacteria and, perhaps, granulation tissue. This finds support in the pathology of chronic bone abscess as explained by Alexis Thompson (5). He studied three specimens secured by amputation and by resection, and describes: a *quiescent stage* in which "the lesion is represented by a minute cavity in the bone, filled with serum, and

lined by a well-defined membrane resembling the periosteum of young bones," the latter developed from granulation tissue which, at the outset had at once walled in the invading staphylococci; a *mature stage* or *stage of abscess*, "the lining membrane becoming again converted into granulation tissue and the serous contents transformed into pus, from which the staphylococcus aureus can be isolated"; and a *stage of perforation* and external discharge. The last is not often encountered. Thompson explains fluctuations in the pain in the "stage of abscess" by variations in the relative production of pus and erosion of bone to accommodate it. He describes, however, only an active process and makes no reference to abscesses that have become sterile.

If the onset of pain signifies the recent formation of pus then the case above reported should be regarded as having been, at the time of the operation, a *subacute*, rather than a *chronic* abscess. It seems reasonable to say, also, that this patient's infection might have developed into a very active process or subsided, leaving a chronic, sterile abscess.

REFERENCES

1. Brickner, Walter M. Chronic medullary abscess of the long bones. Its clinical and roentgenographic features: its treatment by simple trephining. Ann. Surg., 1917, lxv, 483.
2. Bevan, Arthur Dean. Brodie abscess. Surg. Clin. Chicago, 1919, June, 743.
3. McWilliams, Clarence. Central bone abscess. Brodie's abscess; chronic suppurative osteomyelitis. Ann. Surg., 1921, lxxiv, 568.
4. Bancroft, Frederic W. Two cases of localized abscess (Brodie's abscess). Surg. Clin. North America, 1921, i, 1774.
5. Thompson, Alexis. Observations on the circumscribed abscess of bone (Brodie's abscess). Edin. M. J., 1906, n.s. xix.

REPAIR OF TENDONS IN THE FINGERS

By STERLING BUNNELL, M.D., San Francisco

ONE of the most baffling problems in surgery is to restore normal function to a finger in which the tendons have been injured. When we consider that the majority of industrial disabilities are from injuries of the hands, and that a successful repair of an injured finger tendon is a rarity, it is apparent that there are especial attendant difficulties, which cannot be overcome by the ordinary surgical procedure.

Before considering these difficulties and formulating methods of overcoming them, let us think of the hand as a marvelously functionating piece of live machinery and regard it with due respect. Though its tissues are tough and compact, its tendons, tendon sheaths, pulleys, joint surfaces and flexible joint capsules all glide on each other so easily and smoothly that there is little in the refinement of motion that the hand cannot do. If infection or trauma insults these perfected parts, a tissue reaction will occur, which will bind the gliding surfaces together, thicken and stiffen the joint capsules, congeal the tissues of the finger, strangulate the nerves and vessels, and loss of function will result.

Atraumatic technique essential. Surgical trauma so brutalizes the tissues and lowers their vitality that they are an easy prey for the few bacteria that even in spite of careful asepsis are unavoidably present. In most repairs of finger tendons free grafts are put in and as these have such a low resistance to infection, it is imperative that in these cases we use not only superasepsis, but also an atraumatic technique. If we are to avoid binding adhesions, we must not have infection even in its milder form of inflammation, in which no pus is formed, nor must we have aseptic traumatic inflammation, as this reaction to pure trauma is almost as binding to movable parts as is the fibrosis of infection.

As binding fibrosis is our arch enemy and trauma is both its direct and by predisposing to infection its indirect cause, we must if we

are to achieve success in repairing tendons of the fingers adopt an atraumatic technique.

Handling of the tissues should be reduced to a minimum and really to do this is surprisingly difficult. Every time the tissue is touched a traumatism results. If we keep the delicate histological structure always in mind, we will realize the necessity of never pinching or pulling the tissues to the degree of even microscopic tissue strain. The most gentle retraction and sharpest knife-edge dissection is not showing too great a respect for tissues, even if they are at some distance from the tendon. When handling a tendon or sheath, never should even a tiny scratch be made in the delicate membrane covering their surfaces, as here adhesions will form and prevent gliding. If we operate in a field rendered bloodless by the use of a blood-pressure band as a tourniquet, we may avoid the unnecessary trauma of sponging and can by clearly seeing the tissues dissect with exactness and a minimum of traumatism.

In a former paper[1] I have endeavored to formulate the methods and principles whereby we will meet with better success in reconstructing damaged movable parts.

Reconstruction of gliding mechanism. A tendon alone in the tissues cannot functionate as it will be bound by adhesions. To permit movement back and forth a tendon must have in addition a gliding mechanism. To Dr. Leo Mayer[2] and to Professor Biesalski is due the credit of giving us the exact anatomy of tendons and their gliding mechanisms, and the physiological method of transposing tendons, especially in the foot. They have shown how a tendon in the straight part of its course is surrounded by a loose fatty tissue called paratenon which is pulled back and forth in its central zone with the movements of the tendon, and that wherever a tendon turns a corner it runs in a true sheath, in which the surface of the tendon (epitenon) slips freely in contact with the synovial lining surface of the sheath. In other parts of the body a bloodvessel-bearing mesotenon is present throughout the length of the sheath, much like a mesentery, but in the fingers this is absent, being reduced to the two strands of tissue known as ligamentum breve and ligamentum longum.

Fig. 1. Case B. F. P. Thumb on August 19, 1919, was cut half way around its base on the volar side by a bursting bottle, severing flexor tendon. Tendon sutured by his physician 12 hours later. Came for repair March 15, 1920. Power of flexion of last joint of thumb entirely absent.

Operation. Flexor tendon was found to be densely adherent for 1½ inches, united and twice its normal diameter. The adhesions and scar tissue were excised, the tendon was pared down to its normal diameter and a free graft of specialized fat was taken from the paratenon over the triceps tendon and planted sleeve-like about the tendon in the thumb.

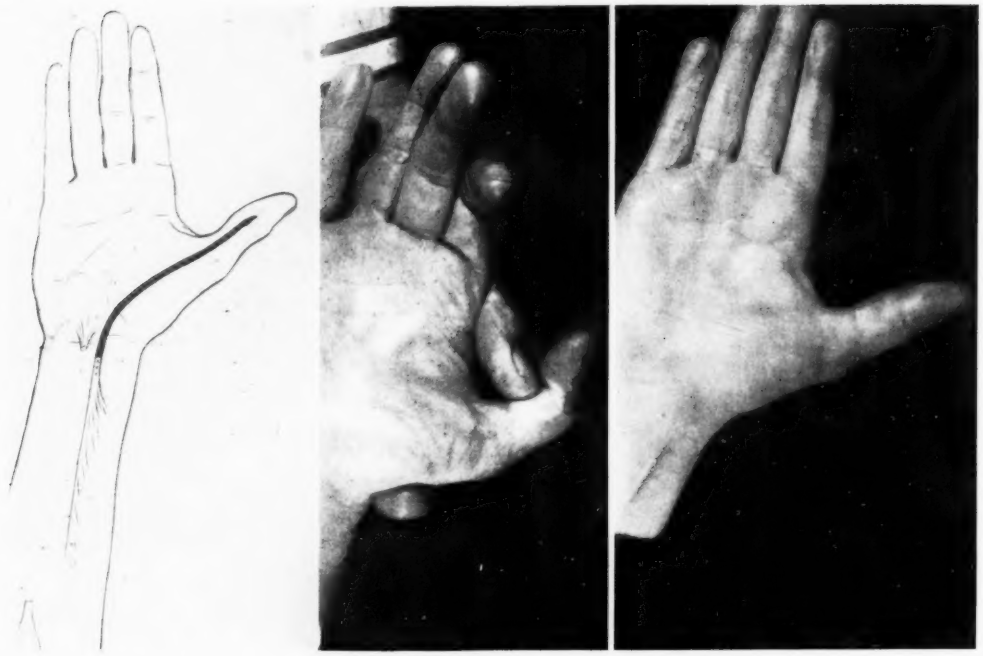
The double photographic exposure shows the degree of function obtained August 9, 1920.

In repairing an old injury of a flexor tendon of a finger it is not practical to suture the tendon within the finger. In the gap between the tendon ends the sheath will have become obliterated and there is not room in a finger to envelope the suture line with a sufficiently thick free graft of fat to allow motion. The exception to this rule is where the tendon is severed in the less usual site opposite the middle phalanx or distally, where the repaired portion need pull through but one joint, in which case a fair result can be obtained. Where the suture line pulls through two joints, it is necessary to remove entirely the damaged tendon from the finger and to replace it with a free tendon graft. The suturing of the tendon can then be done at its insertion and in the palm or forearm where adhesions will do less harm.

If the sheath of the tendon has been damaged, the new tendon will not slip and it will

[1] An essential in reconstructive surgery — atraumatic technique. California St. J. Med., 1921, May.

[2] The physiological method of tendon transplantation. (Extensive bibliography.) Surg., Gynec. & Obst., 1916, xxii, 182.



a *b* *c*

Fig. 2. Case Mrs L. F. Two months previously the flexor tendon was severed at the base of the thumb on a piece of crockery, resulting in complete loss of power of flexion of the distal joint of the thumb.

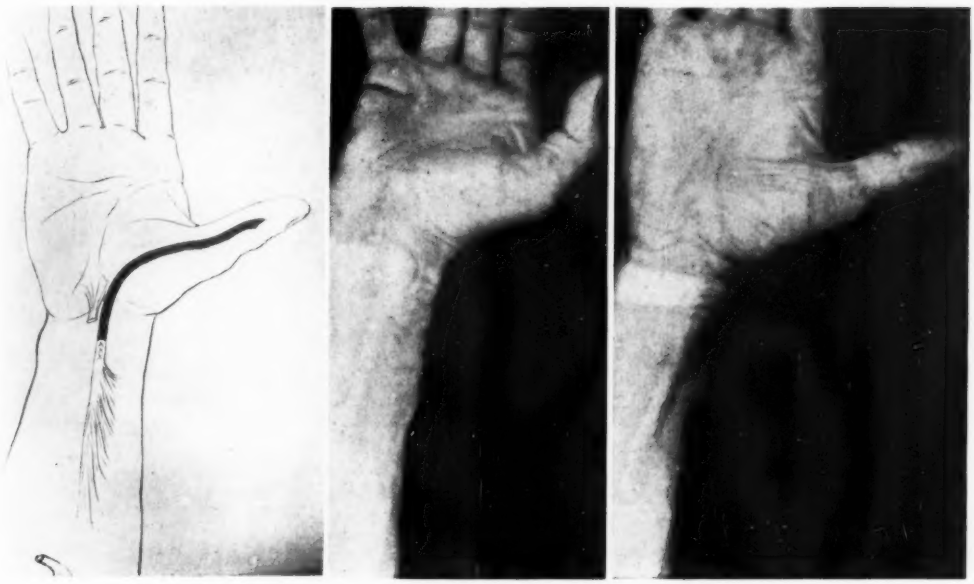
Operation. The proximal end of the flexor tendon was, with the aid of strippers, withdrawn through an incision in the forearm and cut off at the wrist. The distal end of the tendon was removed through an L-shaped incision in the thumb. A free graft of the tendon of the palmaris longus was used to bridge the gap, as shown in *a*.

b and *c* taken 6 months later show degree of function restored to thumb.

be necessary to graft not only a tendon but a gliding mechanism complete. This means a tendon plus its sheath or a tendon plus its paratenon. The placing of both a sublimis and a profundus flexor tendon in the finger is too complicated. It is found sufficient to sacrifice the sublimis and use the profundus tendon only. The prevention of adhesions by the use of foreign bodies, as cargile membrane, etc., has long since been discarded, nor can a silk thread act as a substitute for a tendon in a finger.

Free tendon grafts cause less reaction than foreign bodies. They subsist on the surrounding lymph until they obtain their own blood supply. Often I have uncovered these grafts at short and long periods afterward. At first they show a slight swelling, but later show their original size and appearance and practically are none the worse from the transplanting. Some have even lived through infection. Function keeps a tendon in good condition, just as with bones, and a tendon with all its blood supply, if left without an insertion, will become yellowish and friable.

Sources of free tendon grafts. Grafts are easily obtained from the sublimis tendon, the palmaris longus, the long extensors of the toes, tubularized triceps, fascia lata, and many other sources. If the sublimis tendon of the same finger is used its upper end may be detached through a transverse incision, 1 centimeter long, in the forearm. It is well here to attach the distal end of the sublimis muscle to the profundus tendon for added strength. The incision is short and transverse for cosmetic reasons and to make a shorter distance for adhesions to form along a tendon in the



a *b* *c*

Fig. 3. Case W. Z. Seven months previously, while opening a glass jar, the flexor tendon was severed at the base of the thumb. Power of voluntary flexion of the distal joint of the thumb was completely lost and that of the proximal joint was partially lost, greatly hindering his work as a cook.

Operation. The proximal portion of the tendon was found to be coiled up in a rounded mass an inch above the wrist and to have become yellowish and friable. After uncoiling the tendon and removing most of it, and also the distal end of the tendon still in the thumb, a 5-inch defect in the tendon remained. This was bridged with a free graft from the palmaris longus, including the tendon plus its paratenon, as shown in *a*.

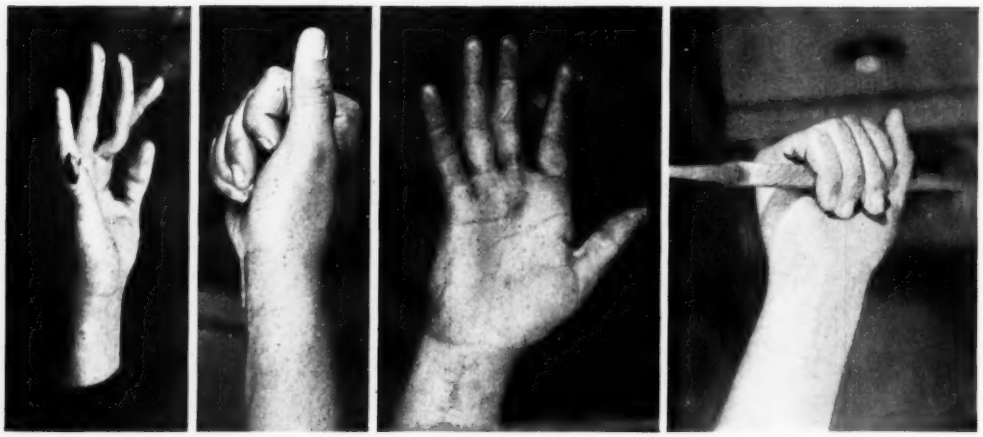
b and *c* taken 11 months later show the degree of lost function restored to the distal joint of the thumb.

The proximal joint functionates normally, though not flexed in the photograph.

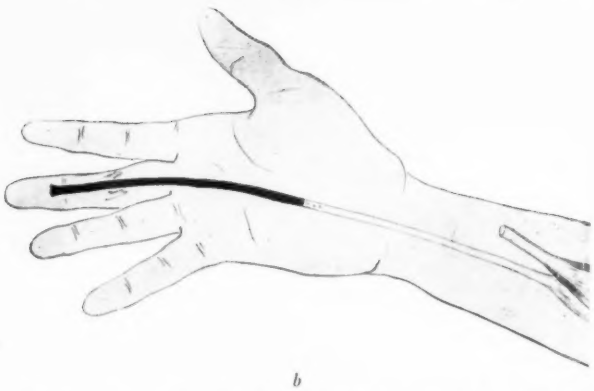
forearm. If the palmaris longus tendon plus its paratenon is used, it will be necessary to make a longitudinal incision the full length of the graft. A long extensor tendon of the toes plus its paratenon in the dorsum of the foot can be taken without getting toe-drop, as the extensor brevis digitorum will serve to extend the toes. If a very long-branched tendon be desired, the extensor tendons to the second, third, and fourth toes can be used. They converge to form the inner tendon of the extensor communis digitorum above the annular ligament and can be severed here and at the toes. The outer tendon of this muscle (to the fifth toe and peroneus tertius) and the tibialis anticus will prevent foot-drop. It is necessary, however, to strip off the paratenon in the foot before these long tendons can be withdrawn.

If the paratenon is not removed intact with the tendon graft, paratenon fat may be gathered from elsewhere and planted sleeve-like about the tendon graft or other tendon to be mobilized. The fat over the triceps tendon is well suited for this purpose. Paratenon fat is specialized fat and differs much from the tough, short-fibered, subcutaneous fat. It is elastic and pliable and can easily be drawn a long distance back and forth from its moorings, as can be seen by comparing the mobility of the skin over the triceps tendon with that over the outer side of the arm.

Method of suture. The suturing of a tendon must be very strong, as it must stand the strain for the 5 weeks necessary for the physiological union to be complete. The stronger the suturing, the more forceful and continuous can be the all-important, postoperative exer-



a *c* *d* *e*



b

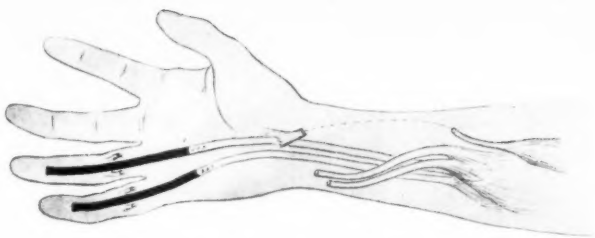
Fig. 4. Case Miss R. K. Four years previously the flexor tendons of the long finger had been severed with a knife over the proximal phalanx, resulting in complete loss of voluntary flexion in the last two joints and most of that in the proximal joint and in the deformity shown in *a*.

Operation. Through an incision in the wrist the sublimis and profundus tendons of the long finger were freed with a stripper to the far end of the palm, where they blended to scar tissue, and were drawn out of the arm. Through an L-shaped incision in the distal crease of the finger the distal end of the profundus tendon was stripped down through the finger until it tapered out. It was withdrawn from the finger and cut off. The sublimis tendon was cut off high in the forearm. With the expanded probe a channel was dilated in the finger and the free graft of the sublimis tendon was drawn through and used to bridge the gap in the profundus tendon, as shown in *b*. *c* and *d* taken 4 months later show the amount of function restored to the finger.

cise. The suture must be braided into the tendon for quite a distance and as little as possible of it exposed to the surface. The knots should be buried. If sewed well, the tendon will break before the suturing will. Any crude handling of the tendon, such as by grasping it with tissue forceps, will result in fraying of the tendon and in scratching and tearing the delicate covering or epitenon, and binding adhesions will result. Therefore, I use a specially devised fenestrated clamp and sew through the slits, making an even machine-sewed union and inflicting minimum trauma to the tendon. This I have described in a former paper.[1] Short tendon grafts may be threaded longitudinally on the two strands of thread. The suture material should be soft, slender, and strong. Pagenstecher is too harsh and will sometimes be extruded. Turner patent ligature silk Nos. 1 and 2 is satisfactory and often on exposing the union months later the suture line and silk cannot be seen. A good method is to let the suturing embrace only the half of the diameter of the tendon away from the mesotenon, so that at least the other half of the diameter of the

[1] Repair of tendons in the fingers and description of two new instruments. Surg., Gynec. & Obst., 1918, xxvi, 103.

a *c* *d* *e*



b

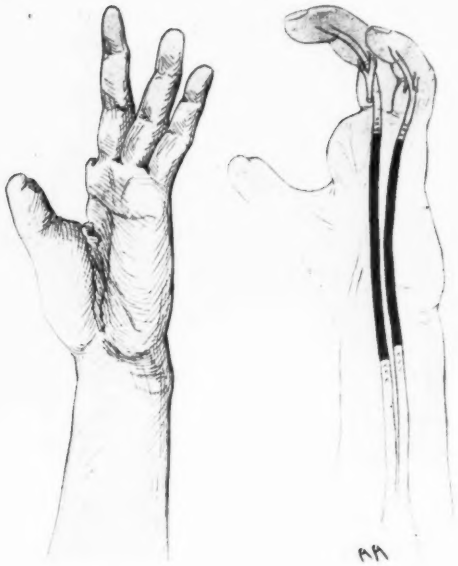
Fig. 5. Case Miss M. D. C. Three months previously, a knife severed the flexor tendons of the ring and little fingers over the middle of the proximal phalanges. In *a* is shown the limit of voluntary flexion in these two fingers.

Operation. All of the flexor tendons were removed from the ring and little fingers. It was found that much of the tendon sheaths of each finger had been destroyed. As substitutes, free tendon grafts plus paratenon fat were used. In the ring finger a sleeve of paratenon fat taken from the forearm was sutured in place and the sublimis tendon of this finger was cut off high in the forearm, drawn through this paratenon sleeve, and used as a free graft to bridge the deficiency in the profundus tendon. In the little finger, the tendon of the palmaris longus plus its paratenon was used to supply the defect in the profundus tendon. Its sublimis tendon was cut off. All tendon suturing was done in the palm and in distal ends of the fingers, as shown in *b*. *c*, *d*, and *e* taken 3 years later show practically normal function restored to hand.

tendon may be unhindered by suture material from making a perfect union. If the union be made in a region such as the forearm, where adhesions are not of such importance, it is well to overlap the cut surfaces of the tendon ends, so as to attain earlier and stronger union for early exercise. The proper tension for uniting a tendon has been worked out by Dr. Leo Mayer, as follows: When the origin and insertion of the muscle are approximated as near together as possible the tension of the tendon should be zero. There is an exception to this rule, however, in the case of a muscle which, owing to the severance of its tendon, has long been contracted. If the rule be followed in this case, it will not be possible to extend the muscle sufficiently for full function, and it will be necessary to make our tendon graft a little longer.

Removing and replacing tendons. It is difficult to remove a tendon neatly from a mass of adhesions and especially from beneath the annular ligament and from within a finger. These are the places where infection does the most damage, as here the firm structures prevent swelling and ischæmia results, encouraging the ravages of the infection. For this purpose I use a stripper specially devised from a cork borer (see former paper) that with a sharp edge and twirling motion planes the tendon smoothly from its binding adhesions. Some of the strippers have flexible handles to fit the curve of a finger.

It is difficult also to place a tendon, with its paratenon, through a tunnel in the finger. This can be done with a flexible probe expanding flatly at its center, so as to widen the tunnel, and terminating posteriorly in a funnel



a *b*

Fig. 6. Case C. H. C. Fourteen years previously a buzz saw cut partially across his hand, entering the cleft between thumb and index finger and severing all of the flexor tendons of the fingers. A severe infection resulted, during which time all of the severed tendons sloughed out from the palm. The index finger had been amputated, joints of long finger were ankylosed and ring and little fingers bent backward at metacarpophalangeal joints. There was complete loss of power of flexion of the fingers, as shown in *a*.

c *d* *e*

Operation. After amputating the ankylosed long finger and excising much of the scar tissue from the palm, the tendon ends were found at the bases of the fingers and in the wrist. Four good tendons were found in the forearm. Two of these were cut off and used as free grafts to join the other two tendons in forearm to ends of flexor tendons in bases of ring and little fingers as shown in *b*.

Six months later the palm was re-opened. The tendon grafts were in good condition and the fat in the palm had improved. The flexor tendons were freed of adhesions and stripped down their full length to the finger tips. The finger joints were forced into full flexion. As the operation was being done under brachial plexus anæsthesia, the man could then flex his fingers well. Active motion postoperatively was persisted in.

In *c*, *d*, and *e* taken 3 months later is shown sufficient function to allow him to return to work for the first time in 15 years.

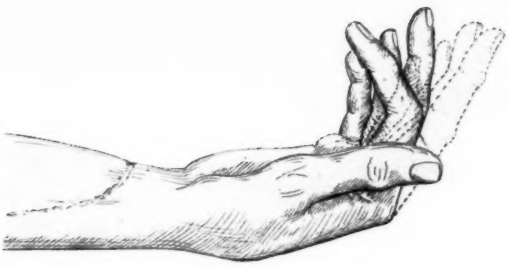
to receive the end of the tendon and draw it through. The sutures from the end of the tendon hook over a peg in a slit in the funnel. A thin rubber tube may envelope the paratenon until it is evenly placed in the finger, and then the tube can be withdrawn.

Freeing adherent tendons. Quite often after a tendon repair or an infection, or even after a general traumatism to the hand or a simple fracture of a phalanx, it is found that the joints can be passively, but not voluntarily, moved because of adhesions between the tendon and the surrounding tissue. If these adhesions are planed off from the tendon by a tendon stripper (mentioned above) and active motion be started at once and persisted in, a great gain or even the normal range of motion will be attained. If the adhesions are light, this may be accomplished through an incision in the forearm. The finger joints are forced into full flexion, and the tendons in the forearm are separately pulled, until suddenly the adhesions yield and full flexion of the finger can be made by pulling the tendon.

Often after a tendon repair it is necessary to do a second operation of freeing the tendon from adhesions. This is best deferred, however, for several months until the original postoperative induration has passed away, otherwise a summation of postoperative induration might defeat our purpose.

Incisions. The selection of the proper incisions is very important. Many fingers are permanently crippled by the median longitudinal incision. This cuts the pulleys, roughens and causes adhesions on the friction surface of both tendon and sheath and leads later to a contraction like that of Dupuytren. If the median longitudinal incision of the finger, whether on the dorsal or palmar sur-

b *c* *d*



a

Fig. 7. Case Mrs. J. M. A year previously she fell through a window severing most of the tendons and the median nerve in the right forearm. Two operations had been performed, uniting the tendons and nerve. Infection had taken place. There resulted a total loss of function of the hand. The thumb assumed the lateral position and had no power of flexion. Limits of voluntary flexion and extension of the fingers are shown in *a*.

Operation. The median nerve which had united was dissected from the dense scar that bound it. The tendons which were all held firmly in scar tissue were dissected free. It was found that the flexor pollicis longus was missing, so the tendon of the flexor carpi radialis was then united to its distal end in the wrist. Several tendons were also missing, including the profundus of the ring and little fingers. The sublimis of the index finger was, therefore, transposed to connect with their distal ends. It was found that the index sublimis had been joined to the distal end of the flexor carpi radialis and that the flexor carpi radialis had been joined to the sublimis of the long finger and that various other tendons had been sewed to the wrong ones. The effect was much like a splicing in a rope. This tangle was corrected by severing and re-uniting the tendons. Then for each finger in turn all of its joints were forced into full flexion and the respective tendons were pulled until the adhesions in the finger gave way. All of the fingers could then be brought into full flexion by pulling the tendons in the forearm. Wrist was then forced into flexion and held so by reversed cockup splint. Thumb maintained in a position apposing the hand by spiral traction. Active postoperative motion was persisted in.

b, *c*, and *d* taken a year later show result. She was able to work and write a legible hand.

face, through the matrix or in the pulp, whether for infection or repair, be eliminated entirely from the surgeon's activities, many fingers will be saved from being permanently damaged. Even in the palm the longitudinal incision leads to contraction. It is far better to make transverse incisions in the palm in the line of the creases or L-shaped ones than longitudinal. In the finger lateral incisions between the two lateral nerves and arteries should be made. They should be interrupted so as not to cut the pulleys. As the pulleys are located at the center of the segments of the finger, the lateral incisions should be opposite the joints. Distally, near the insertion of the tendon, the incision may be L-shaped, one arm running transversely across the volar surface of the finger. It is very important not to cut the nerves of the finger,

a

b

c

Fig. 8. Case Mrs. C. H. Showing method of correcting a flexion contracture of a finger preparatory to a tendon repair. *a*, Flexion contracture. *b*, Flexion contracture excised. Finger hyperextended on a previously prepared and boiled metal splint. Whole thickness free graft of skin from abdomen applied. *c*, Result.

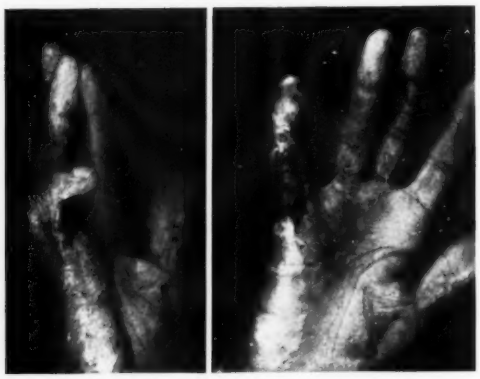


Fig. 9. Case R. W. (colored). *a*, (at left) Flexion contracture from gasoline burn. *b*, Same, after excision, hyperextension on splint, and skin graft by tubularized pedicle graft from side of chest.

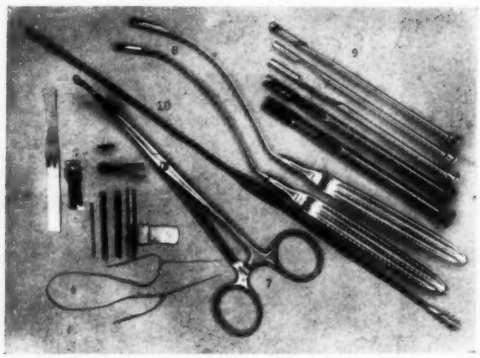


Fig. 10. Special instruments used in repairing tendons. *1* to *7* shows clamp used for suturing tendons, by which a perfect union may be obtained with minimum trauma.

1, is a punched-out matrix band of German silver, gauge .003, detempered at its center by passing through a flame. It is bent, as in *4*, by bending it about a rod and sliding over its two ends a sheath, *3*, as in *5*. In suturing the tendon the clamp is placed about the tendon end and made to hold it firmly by sliding the sheath up snugly and backing it up by a hæmostat, *7*, to be used as a handle. An 8-inch thread, *6*, with two needles is braided through the tendon by sewing through the slits of the clamp. The stitch is started by one needle entering just proximal to the clamp and running diagonally through the tendon and out the next slit. It is then made to encircle some of the tendon fibers and is again thrust through the tendon and out the next slit. At the third stitch the needle emerges from the end of the tendon. Starting at the other side of the tendon the other needle is similarly passed, so that both threads emerge from the end of the tendon. These are pulled firmly to take up all the slack, the clamp is removed, and the other tendon end receives its suture in the same manner. The threads of one tendon are tied each to those of the other, thus joining the tendons. The knots are buried between the tendon ends

Strippers are shown at *9* for freeing, with their sharp ends, the tendon from its binding adhesions. The tendon is held taut and the stripper is passed along it with a twirling motion, planing as it goes. Some have malleable handles, as *8*, for following the curve of a finger.

The flexible probe, *10*, is passed through the finger and its expanded center widens the channel for the reception of the tendon. The sutures from the end of the tendon are caught over the hook in the funnel at the tail end of the probe, so that the tendon end is drawn into the funnel and is drawn after the probe into its future bed.

either in the finger or in the palm, as trophic changes will result. On the extensor surface, crescent or L-shaped incisions are satisfactory. It is important to have the skin incision remote from the tendon graft and tendon suture, as otherwise adhesions and infection will be encouraged. If the pulleys have been destroyed by former ill-advised incisions, they may be reconstructed by fascial grafts. Otherwise the tendon will bow across the flexed finger and loss of function will result.

Flexion contracture of a finger. Flexion contracture of a finger is quite common, due either to suppurative tenosynovitis, or to the ill-advised median longitudinal incision. Mild forms may be corrected by using slow traction with a splint. A wire splint resembling a large safety pin, with two flat ends is laid longitudinally on the volar surface of the finger and with a web strap and buckle looped around each wire, the convexity of the finger is day by day gradually drawn forward between the wires until the finger is hyperextended. This position is maintained until the tissues of the volar surface of the finger have grown long enough to allow correction to be permanent.

Greater degrees of flexion contracture can be corrected by excising the contracting band of skin and subcutaneous tissue and after forcing the finger into overcorrection closing in the denuded area by a tubularized pedicle flap, prepared 10 days previously. Wolfe grafts may also be used, but they necessitate perfect immobilization. Thiersch grafts do not furnish enough subcutaneous loose tissue to make a bed for the later tendon repair. After straightening the finger hyperextension must be maintained for a month by splinting. If the contracture is too extreme, gangrene may result from sudden forced extension.

Indications and contra-indications. It is a mistake to attempt to mend a bone, correct a

contracture, or mobilize a stiffened joint in a finger at the same time that a tendon is repaired, as failure is sure to result. The added trauma will create adhesions to the tendon and also we will have the impossible double indication both to immobilize and move the finger. Unless the joints are freely movable, it is useless to repair the tendon, as it will not be able to move a stiff joint. The adhesions will then catch the tendon at rest and bind it in its bed.

If the joints of a finger are permanently stiffened, it is better to amputate the finger than to try to reconstruct it. Such joints have adhesions between their articular surfaces that will not allow movement, even after all the tissues, including the capsule of the joint, are cut away. If the stiffened joints are not permanently damaged, they may be drawn into the position of flexion by slow, mild, continuous traction on the ends of the gloved fingers. They will then become more easily mobilized, and the new tendon to be put in will have a chance to functionate. Intermittent strong forcing of the joints only results in straining them, which stiffens them all the more. Traction must be mild and kept up for a long time, so that the tissues will have time to grow enough to be relaxed in the new position, not merely stretched.

When the storm of an infection has passed through a finger and destroyed the flexor tendon, there often results a thin, red, shiny finger, with stiffened joints and firm congealed tissue. The contracted scar tissue throughout the finger has strangled the nerves and vessels, so that great trophic changes have resulted. Such a finger is unfit for repair of the tendons, and amputation is to be preferred. For a tendon repair to be worth while, the bed through which the tendon is to be placed must be soft and succulent. It may first be advisable to excise the damaged firm tissue and replace it by means of a tubularized pedicle skin-graft before tendon repair is done. To avoid latent infection and to await the softening up of the tissues, at least 4 months from the time of infection should elapse before a repair is attempted. A freshly cut tendon may be repaired primarily if an excision of the damaged tissue is done, but if over 24 hours have elapsed since the injury, infection will probably result if the tendon is operated on. One had better treat it conservatively and later do a secondary repair.

It is well to beware of an annular scar, or the too close proximity of another scar, as in such a case necrosis is prone to occur from the diminished nerve and blood supply.

There are certain people, and not always old ones, whose joints and tissues have a tendency to stiffen if traumatized or immobilized and these make poor subjects for tendon repair. The presence of osteo-arthritis suggests this type. There are others who have not enough character to withstand the pain of moving their fingers after the tendon repair. With them an otherwise good result may be prevented. Before attempting a tendon repair, one must weigh well the gain of function to be expected with the amount of inconvenience and trouble to the patient.

After-treatment. The after-treatment is all important. It is best to avoid splinting the fingers, as too much immobilization allows adhesions to form. Instead, the wrist can be strongly flexed for a flexor tendon and held so by a check-rein of adhesive plaster running up and around the forearm. The muscles that pull the tendons are thus robbed of their power of too much traction and still they can keep the tendon actively moving. Motion should be encouraged from the first, but too much or too vigorous early motion causes hæmatoma and the opening of incisions. Later, a ball, a rounded match box, or a piece of rubber hose can be carried in the pocket and used for exercise in grasping. The restricting check-rein should not be removed for 5 weeks when union will be firm. Later the galvanic current may be used to contract the muscles sharply, thus snapping tiny adhesions. If the hand is soaked in hot water previous to exercising, it helps considerably. Actual hard work and the lapse of several months finally complete the loosening-up process. Co-operation by the patient is essential, and he must be willing to take some punishment in exchange for a good result.

It is well to give a guarded prognosis in these cases, but it is usually possible to restore from 80 to 95 per cent of lost function.

DEPARTMENT OF TECHNIQUE

POSTERIOR DISLOCATION OF THE FOOT WITH POSTERIOR MARGINAL FRACTURE OF THE TIBIA AND USUALLY WITH A POTT'S FRACTURE OF THE FIBULA[1]

By T. TURNER THOMAS, M.D., F.A.C.S., Philadelphia

TWENTY-FIVE years ago, as an interne, I had a patient with a posterior dislocation of the ankle in which the X-rays showed a posterior marginal fracture of the tibia. The records are not now available but my memory is clear as to the lesion and the difficulty in maintaining the reduction of the dislocation. I lost track of the case but the outcome must have been bad. Figure 1 represents a case concerning the history of which I know nothing, but it probably will answer for the final result in the above case. About five years ago I saw with Dr. Samuel Ellis, of Philadelphia, a woman with what I interpreted as the same condition but in which an X-ray was refused. As in the first case the reduction of the dislocation was comparatively easy, but it recurred as soon as the force was released. Having now what seemed a satisfactory conception of the condition, after reducing the dislocation I merely held the foot in forced flexion and applied a plaster cast. This was removed at the end of 8 weeks and the patient permitted to walk on the foot. Several years afterward Dr. Ellis reported that she had never complained of the foot afterward.

In the past three years I have seen and treated six similar cases and two others of what seems to me an equivalent condition in adolescence. In them the displacement occurred not at the ankle joint but at the lower epiphyseal line of the tibia with a posterior marginal fracture of the diaphysis. In one of these two there was associated a Pott's fracture, in the other not, but there was a Pott's fracture in five of the six dislocation cases. The X-ray illustrations presented here make a detailed report of each case unnecessary as they tell the essential parts of the story, i.e., the positions of the fragments before and after reduction. So far as the patients can judge, the results have been perfect in every case except two recent ones which promise as much as the others have attained, and in that illustrated in Figure 2, 7, in which early reduction was not permitted.

Stimson says of this condition: "I have never seen it in a fresh injury and do not know whether it could be recognized." I have assumed the presence of the posterior marginal fracture of the tibia whenever I found the posterior dislocation at the ankle joint, except in the first case seen in my interne days. In the second case it was assumed to be present but the X-ray was refused. In every other case the X-ray later showed this fracture to be present. In the third case, because the accident happened in the evening after the X-ray laboratories had been closed, the fracture was reduced and a cast applied before the X-ray was taken.

It has long been held that there is a decided tendency, in Pott's fracture, for the foot to slip backward, as one widely used textbook puts it, "sometimes so far that the body of the astragalus lies entirely behind the tibia; and this displacement is frequently overlooked." The Paris Surgical Society, in 1912, as the result of a paper read before it by Quénu, took such interest in this subject that nine of its meetings in that year were given up to its discussion. Quénu said that great physicians, such as Tillaux, have passed their lives insisting that the posterior dislocation of the foot was the result of a Dupuytren's fracture, and they have erred in recognizing only the essential lesions of this fracture, as the fractures of the internal malleolus and of the fibula. From cadaver experimentation, Quénu concluded that when all the essential lesions of a Dupuytren's fracture are present, a posterior dislocation of the foot cannot be produced. He also concluded that a posterior dislocation of the foot indicates the presence of a posterior marginal fracture of the tibia (such as is shown in the accompanying roentgenograms of my cases). Until the discovery of the X-ray it appears that the only authentic reported cases were those found postmortem (Earle, 1828–29; Malgaigne, 1832; and Adams, 1836). Grashey, in 1907, reported finding in 1500 roentgeno-

[1] Read before the Lackawanna County Medical Society, Scranton, Pennsylvania, September 20, 1921.

grams of all kinds of fractures, four "oblique fractures of the posterior tibial margin with posterior dislocation of the foot." Meissner, in 1908, reported a study of this condition based upon 19 cases. He had one case of isolated fracture of the posterior tibial margin, i.e., without posterior dislocation of the foot; 8 cases of the posterior marginal fracture of the tibia and posterior dislocation of the foot, with fracture of both malleoli; 1 case of the posterior marginal fracture and posterior dislocation, with fracture of the internal malleolus; 5 cases of the same fracture and dislocation with fracture of the fibula, and 4 cases with separation of the epiphysis.

Posterior dislocation of the foot may occur without a posterior marginal fracture of the tibia but seems to be very rare. Auvray reported a case before the Paris Surgical Society during these discussions, in 1912, and exhibited the X-rays of it. After examination of them by the members of the society there was manifested a difference of opinion as to whether they showed a posterior marginal fracture of the tibia or not. Quénu said that for one certain case and two disputable cases of posterior dislocation of the foot without a posterior marginal fracture, there was an abundance of roentgenographed cases of this posterior dislocation showing this fracture. Pels Leusden said that it was always present and Meissner that it was always or nearly always present.

What made these discussions by the French surgeons so warm and prolonged was that Quénu was attacking the long established view that the posterior dislocations were not the result of the Dupuytren or Pott's fracture but that the posterior marginal fracture of the tibia was practically always associated with and necessary to it. Although small, my experience offers a substantial corroboration of Quénu's view. Indeed, I assumed this etiology before I looked up any reference in the literature, conceived and applied my simple treatment and obtained good union and function in four cases before doing so.

For some years I have been interested in the mechanism of fractures and the cause of typical deformities, believing that a better knowledge of them would improve our methods of treatment, and in the *Annals of Surgery*, September, 1919, p. 359, I offered a paper on these phases of fractures and dislocations of the upper extremity. We have had ponderous works and much cadaver experimentation on the mechanism of fractures and dislocations, but with the exception of the dislocations of the hip we have made very little progress from them. The only worth-while proof of the value of cadaver experimentation is in the improved results of the treatment based upon it. The results in the cases here reported and illustrated are offered as further proof of the ideas offered in the above mentioned paper. The fall is assumed to be the great cause of fractures and dislocations, but we cannot give direct proof of this because we cannot reproduce in the cadaver the ordinary fall of the living body nor the effect of the fall except in a few instances. In 1905[1] I reproduced fractures of the head of the radius repeatedly and a few Colles' fractures by the same mechanism as in the fall on the hand following von Bruns.[2] In my opinion, there are only two fractures in the body concerning the mechanism of which we talk with anything like confidence and unanimity: the Colles' and the Pott's fractures. We probably all agree that the usual Colles' fracture is due to the fall on the hand and that the usual Pott's fracture is due to eversion or inversion of the foot. At first thought, it might prove a little disturbing if anyone should ask if these mechanisms have been demonstrated by experimentation on the cadaver. Most of us would know little and care less about it. In my opinion, the proof is in the deformity. We have long ceased to attribute such deformities to muscle pull. The lateral deviation of the foot shown in Figure 2, *1*, was obviously caused by a force driving the foot in that direction. It is equally obvious that the posterior marginal fracture of the tibia and the posterior dislocation of the foot shown here were due to a force driving the foot upward and backward. A clear history of such

Fig. 1. Old untreated posterior dislocation of the ankle with posterior marginal fracture of the tibia. *a* (at left), Lateral view. *b*, Anteroposterior view. In *a* can be seen the callus resulting from the tibial fracture extending backward and downward to the posterior margin of the astragalus. A fracture of the internal malleolus and of the postero-inferior margin of the fibula are also seen.

[1] Univ. M. Bull., 1905, Sept. and Oct.

[2] Zentralbl. f. Chir., 1880, vii, 353.

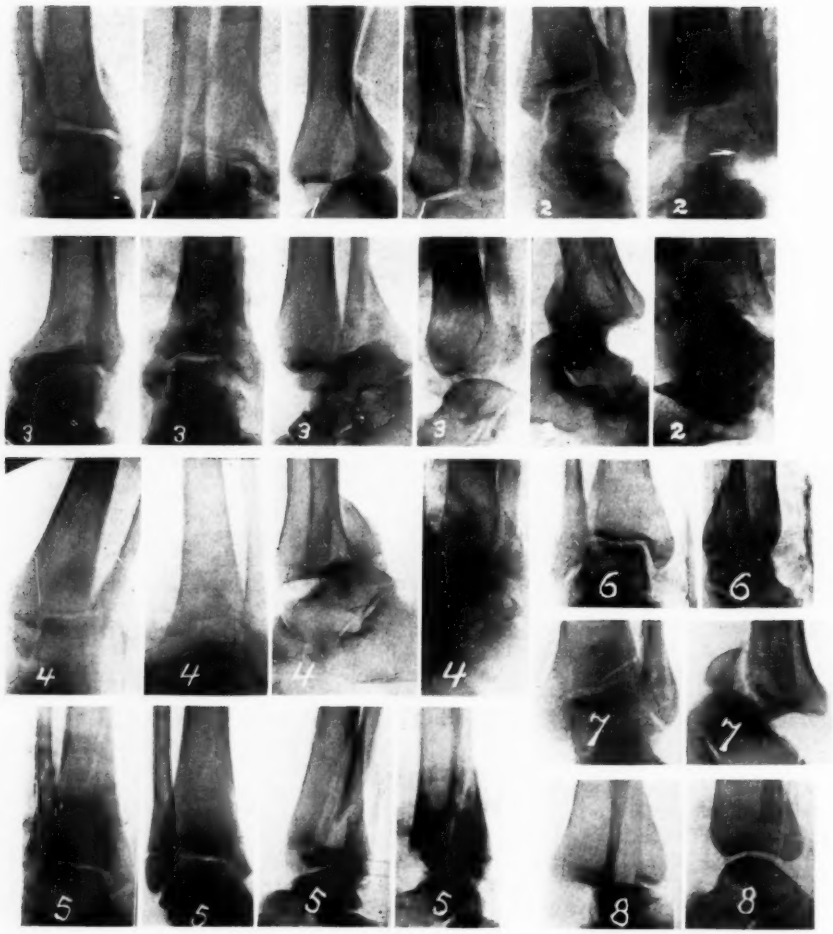


Fig. 2. The cases represented here by *1*, *2*, *3*, *4*, and *5*, show in each instance, the lateral and anteroposterior views before and after the reduction of the displacements. In *6* we have both these views after reduction only, because none was taken before reduction. In *7* the patient refused to permit reduction and in *8* the anteroposterior views taken before and after reduction were lost.

a force was obtained only in connection with the case illustrated in Figure *2*, *6*. We must assume it for the others.

The best method of correcting the displacement is obviously to reverse the fracturing force as indicated by the position of the fragments and drag them back into their normal positions. It has so happened that in every one of these dislocations in which there has been associated a Pott's fracture of the fibula the general anæsthesia has had to be given a second time because, notwithstanding the effort made to correct both displacements in the first trial, the posterior displacement, although very much improved, was not completely reduced in any. During the reduction, first, traction on the foot was made with full strength, then the foot was carried forcibly into dorsal flexion and finally into abduction or adduction according to which was necessary to correct the lateral deviation of the foot. Overcorrection is not likely to occur, but undercorrection not infrequently happens. In Case 3 (Fig. 2,

6), in which there was no Pott's fracture, the first attempt was completely successful. For the second trial in the others, the cast applied in the first, was so cut away as to leave the upper portion down to the tips of the malleoli in order to preserve the correction of the Pott's fracture, and high enough anteriorly and posteriorly to permit the necessary dorsal flexion of the foot on the second trial which was sufficient in every case of pure dislocation-fracture. In the epiphyseal separation with dislocation-fracture (Fig. 2, 5), while the patient was under ether for the second trial at correction of the posterior displacement, an X-ray was taken and developed immediately after the first attempt at correction. This proving unsatisfactory, without permitting recovery from the anæsthetic, a second attempt and an X-ray were made and as the result was still unsatisfactory a third correction and X-ray were made with the result shown here.

It will be seen on close examination of the roentgenograms that there is usually a posterior marginal fracture of the fibula also, even in Figure 2, 6, in which there is no Pott's fracture. In Figure 2, 1, the Pott's fracture is seen to involve the posterior marginal fracture of the fibula. Clearly the posterior marginal fractures are due to the same force as the dislocations and are essential to the dislocation, while the Pott's fracture must result from the lateral deviation of the foot. In Case 3 there was no Pott's fracture because the foot did not turn sidewise.

It has not been considered worth while to furnish individual histories of the different cases except the facts given in connection with the first two. Of others it seems sufficient to say that they all fell and all got perfectly well, except the last who has had the cast removed and when last seen had not yet recovered normal motion in the ankle, and the case illustrated in Figure 2, 7, who refused treatment. He was an excitable and apparently rather irresponsible young man who requested that he be transferred to another hospital than the one in which I first found him. He was sent to another hospital but later refused to be admitted there. This was about 15 months ago and I was told recently by a neighbor that he had since been operated on twice by another surgeon and that he could now walk. Rowlands[1] illustrates two cases by X-ray and one of them by photographs taken before and after operation. In this case before operation "the limb was almost useless." The operation consisted of a tenotomy of the tendo Achillis with osteotomy of the tibia and fibula through a small incision 1 inch above the tip of the internal malleolus. The function of the limb later was good, the patient being able to walk long distances without pain and move the ankle well.

CONCLUSIONS

1. Posterior dislocation of the ankle with fracture of the posterior tibial margin and Pott's fracture has received little attention in this country. The dislocation has been generally attributed to relaxation of the ankle joint resulting from the Pott's fracture.

2. The prognosis will depend largely upon the degree to which the reduction of the displacements has been accomplished and maintained during the development of bony union. Without reduction the impairment of function must be serious. With reduction the function has been essentially normal in all of my reduced cases except in two recent ones which have not yet had sufficient time to recover normal function.

3. The reduction should always be proven by the X-ray, and it should be maintained by a plaster cast because no other method will maintain the reduction satisfactorily. Moreover, for the same reason, the cast should not be removed until bony union is assured.

[1] Brit. M. J., 1919, Dec. 6, p. 735.

A SELF-RETAINING, EASILY REMOVABLE DRAINAGE TUBE

By MAX BALLIN, M.D., F.A.C.S., Detroit, Michigan

EVERY surgeon knows the difficulties encountered after certain operations in keeping drainage tubes in place. I refer to drainage following operations upon the gall bladder, draining the urinary bladder, drainage of deep axillary wounds after breast operations, etc. The generally adopted method is to fasten the tube in the depths of the wound by means of a loose catgut suture, then either forcibly tear this suture when removing the tube or wait for absorption of the catgut. The first procedure is painful and the second cannot always be accomplished at the time desired, because catgut is not absorbed uniformly. On the other hand, if the tube is not fastened at all, it may easily slip when making the final toilet—removing the pads or suturing the wall. Such a loose and unfastened tube frequently does not perform its function for the reason that the end does not remain in its proper place.

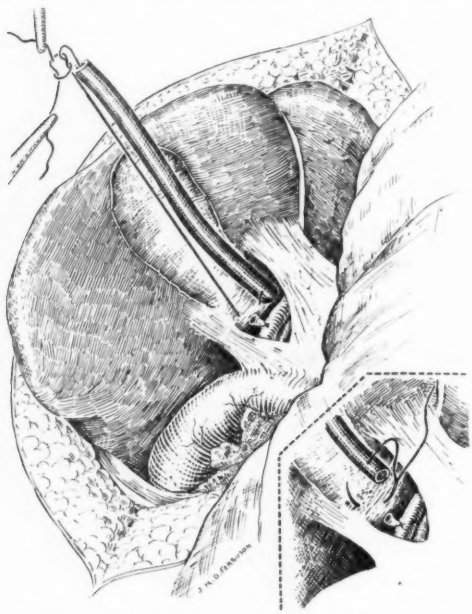


Fig. 1. Drainage tube fastened close to stump of cystic duct after cholecystectomy. The suture, passed through the tube, is fastened close to the stump of the cystic duct as shown in the insert, then tied over the end of the tube, outside the abdomen, without bending the tube.

We have overcome this difficulty (perhaps others have used the same simple procedure) in the following manner: A long black linen thread is drawn through the lumen of the tube, passed through a needle and stitched through the side of the tube close to its end. A stitch is then taken in the depth, fastening this end of the tube to the tissues close to the point where drainage is to be expected (Fig. 1). The thread is then brought out of the wound and tied over the exposed end of the tube. In this way the pads can be easily removed without dislodging the tube.

When it is desired to remove the tube, the thread outside the tube is cut. To preserve asepsis, this is done just beneath the skin after applying a drop of iodine. The tube is then easily withdrawn, the thread being pulled from the tissues without causing any injury or pain.

We have found this tube very practical because of the painlessness of its removal. It can be used wherever retention of drainage tubes in their proper place is difficult. In cholecystostomy we suture the tube to the incision in the gall bladder, tightening the gall bladder about it with a purse-string which does not perforate the tube. In cholecystectomy, as illustrated, the tube is fastened close to the stump of the cystic duct. In prostatectomy we suture the tube to the incision in the bladder, tightening the vesical walls around the tube by a few sutures. In breast amputations we fasten the tube to the stump of the pectoralis muscles.

THE UTILIZATION OF THE PECTINEUS FASCIA IN CERTAIN FORMS OF INGUINAL HERNIA

By WALTER D. WISE, M.D., F.A.C.S., Baltimore

WHILE in some noted clinics, the step of cord transplantation in the repair of herniæ has been abandoned, we have felt more convinced each year that in the majority of cases it is a good procedure. We do not use it in children or in young adults with indirect herniæ, who have well-developed and well-arranged muscles with a thin-walled, narrow sac. It is used in all direct herniæ regardless of the size of the sac; in all recurrent herniæ and practically all patients over 25 years of age suffering with the indirect variety. Formerly it was not done when using a local anæsthetic, but lately the cord has been transplanted in these cases also. After the adoption of the plan outlined, our recurrences were fewer than with the cord left in its bed, but we still had an occasional recurrence, particularly in patients over 30 years of age who had been operated upon for direct hernia. Three years ago, following the lead of A. C. Harrison, we began to use the imbrication method of carrying the edge of the external oblique down to Poupart's ligament along with the internal oblique and conjoined tendon. This is done with a figure-of-eight or double row of interrupted sutures and the edge of Poupart's ligament brought up and sutured to the external oblique with interrupted sutures as described in several recent articles on the subject. The cord is placed between the external oblique and the subcutaneous tissues. In this way, of course, the inguinal canal is closed completely at its medial end. Usually this method of closure makes a most satisfactory repair. We have not attempted to trace our cases, but in the 3 years since its adoption, no instance of recurrence has come to our attention (with the exception of one in which in some manner the, or a. sac was overlooked). The series contains many cases of recurrence after operations by other methods and one of double recurrence after a Bassini operation for double direct hernia, considered inoperable, but operated upon in each instance at the insistence of the patient. In this case there was as great a dearth of structures as is likely to be seen, and the patient was 40 years of age. One side has been operated upon by this method 22 months and the other 18 months,

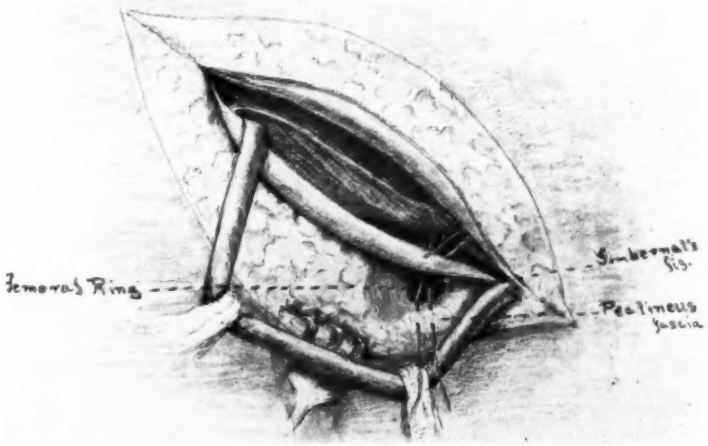


Fig. 1. Showing a figure-of-eight suture beginning and ending by passing through the pectineus fascia.

with no sign at this date, of any weakness in the inguinal regions. All the operations were done by the writer. While we feel that this is, to us, the most pleasing operation so far used and the results satisfactory, yet no possible chance of improving a hernia repair should be neglected. It is this feeling that prompts the suggestion of a modification which is useful in some cases.

While it is remembered that the medial end of Poupart's ligament is re-enforced by Gimbernat's ligament and is, in most instances, strong enough to meet the demands of any hernia operation, nevertheless, it is in some patients weak, frayed, or so thin that it does not seem equal to the responsibility placed upon it. Of course all structures in the repair of a hernia should come together without tension, but this is not always entirely feasible and in the recovery from the anæsthetic and during convalesence there are apt to be periods of strain in which an inferior quality of Poupart's ligament might not hold. In this class of cases, it is our custom to start the figure-of-eight suture in the pectineus fascia and muscle, passing it through Poupart's ligament, catching the conjoined tendon and then the shelving edge of Poupart's ligament, back for the edge of the external oblique, thence through Poupart's ligament again to pick up the pectineus fascia and muscle. It does not change the usual relationship of the structures, but the inclusion of the pectineus fascia and muscle with Poupart's ligament gives a much greater sense of security for the one or two sutures thus placed. In addition to this, it at the same time closes the femoral canal as is done in the repair of a femoral hernia. If it is true that the repair of an inguinal hernia tends in a certain number of cases to the production of a femoral hernia, then these sutures materially lessen this tendency. Obviously the placing of these sutures is limited laterally by the femoral vein, from which point the usual manner of placing sutures is followed.

THE USE OF THE ELECTRIC CAUTERY IN LARYNGEAL TUBERCULOSIS[1]

By GEORGE B. WOOD, M.D., Philadelphia

THERE are few morbid conditions that cause more distress and pain in the final agonies of life than does advanced laryngeal tuberculosis, and there is scarcely another condition in which the physician feels more hopeless not only in his efforts to prolong life but also in his endeavors to alleviate suffering. There was a time when almost every case of laryngeal tuberculosis, incipient or otherwise, held for the afflicted patient a future to which even his physician could look forward with only dread and misgiving. Of recent years, however, a gradually growing confidence has replaced the pessimism of yesterday, so that today the laryngologist is not necessarily touched by panic when an incipient case of tuberculous involvement of the larynx is first diagnosed. For this, he has to thank not only the modern treatment of general tuberculosis, and we must recognize its efficiency, but also the improvements in local therapy. The local treatment has two purposes: one palliative, applicable especially to advanced and hopeless cases, and the other curative. Both of these, however, overlap in their application. Palliative measures will, at times, permit the swallowing of more food with a consequent improvement in the nutrition of the patient, while, on the other hand, the curative methods are frequently, and almost at the moment of their adoption, attended with distinct lessening of the pain and soreness. In this paper, I am dealing only with the question of curing laryngeal tuberculosis. In my experience, the application of medicine, such as lactic acid, formaline, and other germicides has little effect upon the progress of the tuberculous disease, though their use is frequently indicated to combat secondary infections. The real curative measures belong to the domain of surgery. Bloody surgery of the tuberculous larynx, though frequently accompanied by very favorable results, has reached the end of its usefulness for the sole reason that the electric cautery will in almost every instance accomplish the same results, with none of its disadvantages, and it does exceeding more than could ever be hoped for from other surgical procedures. It is undoubtedly the method *par excellence*, and its use brings to the surgeon that peculiar sense of elation which he feels when, through his interference, suffering and death have been averted.

[1] Presented before Clinical Congress of American College of Surgeons, Philadelphia, October 24–28, 1921.

Clinically, laryngeal tuberculosis assumes various forms due to the anatomy of the part involved, to the resistance of the individual, to the presence of secondary infections, and to other modifying circumstances, but the disease itself is essentially a cellular infiltration with the formation of tubercles. This infiltration is avascular so that the cells in the center of the enlarging tubercle lacking nutrition rapidly degenerate under the action of the tuberculous toxins giving rise to the characteristic caseation. If it were possible to revascularize this tissue, a big step would undoubtedly be taken toward the cure of the lesion. Bier recognized this fact when he introduced his method of inducing hyperæmia. I believe that the beneficial action of the cautery is due more to the revitalization of the tissues than to the destructive action of the heat.

In 1910, I removed with a cautery snare, for diagnostic purposes, a portion of a suspicious tonsil from an arrested case of pulmonary tuberculosis. The portion of the tonsil removed showed extensive typical tonsillar tuberculosis. Five days later, I removed another portion of this tonsil, including the scar made by the previous cauterization. In this scar, there was already a well-developed fibrous tissue, changing the histological picture so that now only a single tubercle was found after the examination of numerous sections and this small tubercle was being rapidly converted into fibrous tissue. This finding led to a series of experiments on guinea pigs. After experimenting with tubercle bacilli of various degrees of pathogenicity, I was finally able to produce localized cutaneous lesions of the abdomen. Some of these lesions I partially excised, as controls, and others I cauterized, not destroying them completely, but only making a fairly deep puncture cut through the center with the electric knife. The tubercles, partially excised, developed without any evidence or even retardation, while those cauterized invariably disappeared. By killing the pigs at various stages, a histological study was made of the healing process.

In 3 days after the cauterization, there developed a distinct zone of inflammatory reaction around the area of destruction. This reaction was manifest by the presence of newly formed blood vessels, congestion, and fibroblasts. In 6 days, the zone of reaction was much more marked, and the blood vessels were larger and more numerous. Also, there was a distinct deposit of fibrous tissue between the epithelioid cells of the tuberculous mass. At this time, the tuberculous process beyond the zone of reaction was apparently unaltered, except that it seemed to be decreasing in size. In 20 days, a complete fibrous cicatrix had formed throughout the zone of the inflammatory reaction, and, though there was still some evidence of the tuberculous disease on the tissue beneath the scar, the epithelioid cells were being rapidly replaced by fibrous tissue throughout the entire tuberculous area. To summarize: An eschar formed by the cauterization is sloughed off by the formation of granulation tissue. The development of the granulation tissue is attended by the formation of new blood-vessels which grow through a previously avascular area. This brings nutrition to the fixed connective-tissue elements, enabling them to withstand the action of the tubercle toxin, and it is also possible that the epithelioid cells themselves, because of the added nutrition, develop into fibroblasts. The object, then, of our cauterization of a tuberculous larynx is not so much a destruction as it is a healing of the tubercle, and the great advantage to the patient that the cautery has over bloody surgery is that important organs are not removed or destroyed except by the disease itself. At the present time, I believe that all the various clinical types of localized tuberculous lesions, be they infiltrative, ulcerative, or of the tumor type, if they are within reach will be more successfully combatted by the cautery than by any other form of treatment, no matter in what part of the throat or larynx they occur.

TECHNIQUE

The object of the cauterization is, as has been stated, the production of an eschar rather than destruction of all the tuberculous tissue, and this fact makes the procedure a comparatively minor affair. The application of the cautery may be done either by direct or indirect laryngoscopy. There are a number of laryngologists who prefer the direct method and some who even use suspension laryngoscopy. In certain cases, the direct method can be used with advantage, but the ease with which the cauterization of the larynx can usually be accomplished by the indirect method makes this method the one of choice in the majority of cases. Of course, the laryngologist must be trained to the intralaryngeal manipulation of instruments.

Concerning the equipment, it is important that the supply of electricity, whether from battery or from a street current controlled by a rheostat, is of sufficient strength to heat the cautery knives to a white heat almost instantaneously. Also, the platinum point of the knife should be shorter than the usual pattern in order to prevent burning

of the normal mucosa which is apt to occur from spasmodic closure of the larynx. Three types of knives are necessary. A sharp, pointed one for puncture; a large, flat knife for searing ulceration, and a small, wire loop for treating small infiltrations.

Cauterization of the larynx should always be done under local anæsthesia. I prefer the following method: 1 grain of powdered cocaine is placed in sterile glass and a cotton applicator soaked with sterile water is dipped in the powder. This application is carried into the larynx as far as possible, though on the first trial in many cases the patient's reflexes will prevent the swab being introduced below the ventricular bands. After waiting 3 minutes, a second application is made, using more of the original grain of cocaine, and it will be found at this time that the swab can be carried directly to the area to be cauterized. After waiting another 3 minutes, we can generally proceed with the cauterization. However, sometimes a third application is necessary. The swabbing has, in my hands, been much more efficacious in producing anæsthesia than the instillation of cocaine with the syringe.

Experience alone enables the operator to judge best the amount and character of the burn which he should make in each individual case. As a guiding rule, infiltration and swelling require ignipuncture, while ulcerations should be seared with a broad knife. Tuberculomata, especially those occurring in the interarytenoid space, must be more or less completely destroyed and here, again, the larger knife is necessary.

When the epiglottis is involved without ulceration, a series of punctures should be made into the swelling, perhaps one-eighth to one-quarter of an inch apart. In making these punctures, a sharp-pointed, short knife while still cold is plunged into the tissue, and then the current turned on for a moment until there is a surrounding areola of white eschar. If there is ulceration, the exposed tissue should be seared with a flat, broad knife. Formerly, and even today by some laryngologists, a badly diseased tuberculous epiglottis calls for immediate removal. I believe, however, that better results can now be obtained with the cautery in almost every instance, though freedom from pain may not be quite as quickly obtained. The very grave risk of a dissemination of tubercle bacilli, however, is avoided and we are able to preserve as much of the epiglottis as has not been absolutely destroyed by ulceration.

The use of the puncture for infiltration and the broad knife for ulcerations applies to other portions of the upper larynx besides the epiglottis. In the pyriform swellings of the arytenoid, which are so common in laryngeal tuberculosis, ignipuncture is remarkably efficient, and the rapid reduction in the swelling is astonishing. Below the level of the arytenoids, however, ignipuncture should not be used, except perhaps in cases attended with marked swelling of the ventricular bands and the walls of the larynx just above them. Infiltrative growths below this level, interfering with the functioning of the chords, are usually so intimately connected with these important structures that great care must be exercised in their reduction. The amount of destruction can be more accurately gauged, if the tissue is attacked from the surface than from a puncture of indeterminate depth. In this region, an attempt should be made to remove the exuberant tissue down to the level of the normal structures, and not deeper unless the disease process has already destroyed their contour. When the ventrical of Morgagni is involved, as it frequently is, a broad knife bent sideways should be introduced just below the edge of the false chord. The large tuberculomata which occur in the inner arytenoid region are most efficiently attacked by a broad, large knife. A small wire loop makes the best type of knife for dealing with small tuberculomata on the vocal chords or vocal processes.

I have never seen any very violent reaction following the use of the cautery in the larynx, such as might threaten closure of the larynx from œdema, and usually the resulting soreness is only enough to cause a slight discomfort for the first 24 or 48 hours. In two cases, complete aphonia followed cauterization of the vocal chords; one lasting only for 2 days and the other for an unknown length of time, as the patient passed out of my hands soon after this happened. I cannot recall, at the present writing, any other untoward result, great or small, following the use of the cautery in the larynx.

The repetition of the cauterization should not be undertaken until the previous one has healed, unless an entirely new area of the larynx needs to be treated. Then a second cauterization may take place within a few days as soon as danger of any reaction has passed. The benefit to be expected from a cauterization cannot be fully determined until probably 3 or 4 weeks have elapsed, during which time the slow process of cicatrization is going on. On the other hand, it is not uncommon to see the tuberculous ulcer covered with normal epithelium within a week or 10 days following the cauterization.

There are, of course, therapeutic measures necessary to a successful handling of laryngeal

tuberculosis, other than the use of the cautery, as the local lesion, in the vast majority of cases, is associated with pulmonary disease. It is essential that appropriate general treatment be adopted for the control of the pulmonary condition and this general treatment is an important aid to the local therapy. In many of these cases, however, the cure of the laryngeal condition is so important that sometimes the general therapy has to be modified so that the patient may be in the hands of a competent specialist. Also as to local measures, we must recognize the importance of vocal rest, the application of various medicines for the control of secondary infections, and the relief of pain. Untreated laryngeal tuberculosis usually follows in its course the progress of the pulmonary condition, although frequently it becomes the predominating lesion, developing much more rapidly than the disease in the lungs. On the other hand, I have frequently seen laryngeal lesions heal under the application of the cautery while the pulmonary condition was growing progressively worse. I am so enthusiastic about the use of the cautery in this disease that I believe that 90 per cent of incipient cases can be cured and that the benefit obtainable is only limited by our ability to reach all of the diseased structures, provided the patient has enough vitality to produce the necessary reaction for the healing of the burned area.

Cauterization with the electric knife is not a new method of treating laryngeal tuberculosis, nor in bringing this subject to your attention at this time am I offering any particular technique, but I am simply making a plea for its more universal adoption, convinced as I am of its efficiency in treating this disease. A sanitarium for the treatment of tuberculosis unequipped for the cauterization of laryngeal lesions is not a proper place for these patients to be taken care of and the laryngologist who fails to make use of its therapeutic perfection is also not prepared to handle these cases.

SQUAMOUS-CELL EPITHELIOMA OF THE LIP

Its Surgical Indications

By JOHN HUNT SHEPHARD, M.D., San Jose, California

In 1919 A. C. Broders[1] demonstrated that it is possible to classify squamous-cell epitheliomata, according to their degree of malignancy, into four grades or groups.

Prior to his work pathologists seemed content to report a given lesion of the lip as being "benign" or "malignant." Occasionally one would venture the statement that a certain growth was "highly malignant," basing his opinion upon the degree of cellular activity as manifested by the invasion of the adjacent musculature by the cancer cells. It has been only a few years since medical schools taught that the presence of pearly bodies is essential before making a diagnosis of squamous-cell epithelioma. While approximately 98 per cent of all squamous-cell epitheliomata do show pearly bodies, their presence is not essential; they result from the process of differentiation.

For years it has been known that some lip cancers are fatal while others are not. Most surgeons have had the experience of telling a patient with lip cancer that his only hope for a cure depends upon a radical surgical operation, only later to be embarrassed by learning that his former patient had been cured by some paste, plaster, or other form of non-operative treatment.

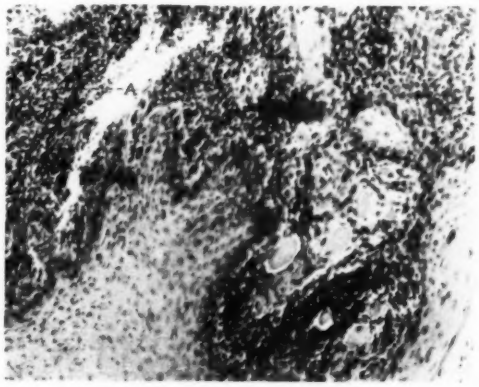


Fig. 1. Squamous-cell epithelioma of the lip, grade 1. Fully three-fourths of the tissue is differentiated. *a*, Leucocytic infiltration.

[1] Broders, A. C. J. Am. M. Ass., 1920, lxxiv, 656-664.

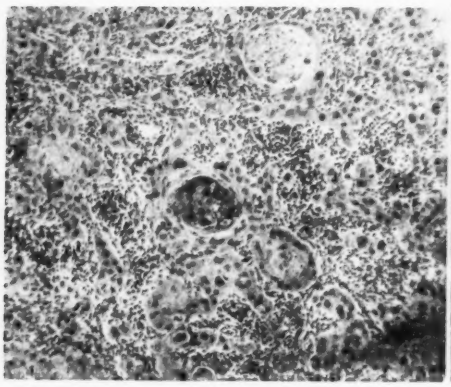


Fig. 2 (at left). Squamous-cell epithelioma of the lip, grade 2. Differentiated and undifferentiated tissue about equal.

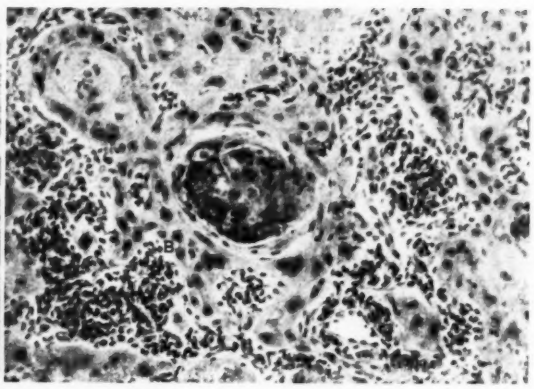


Fig. 3. Squamous-cell epithelioma of the lip, grade 2. *b*, "One-eyed cell." Differentiated and undifferentiated tissue about equal.

The indications for operation of lip cancer have, in the past, been based upon clinical judgment and individual experience. Surgeons have had no common ground upon which to compare their operative results. Some have advocated a complete dissection of the submental and submaxillary triangles in every case; others advising such dissection only in those cases presenting palpable glands in the triangles; others limiting their operative work to the immediate vicinity of the growth while the most radical have advised a complete block dissection of the entire side of the neck corresponding to the side of the original growth together with the removal of all gland-bearing tissue from the opposite submental and submaxillary triangles.

Broders found that the growths comprising his four grades or groups possess a definite and different killing power. "Among the known causes of death, deaths from epithelioma were as follows: none in grade one; 54.9 per cent in grade two; 84.21 per cent in grade three, and 100 per cent in grade four." These results were irrespective of the type or extent of treatment. He also found that all patients showing metastasis in the cervical lymph nodes or in any two or more groups of lymph nodes, at the time of operation, had died and that of the patients reported dead who had shown metastasis over 90 per cent had died from epithelioma.

We note that those cancers of the lip which fall in grade one and which comprised 15.82 per cent of Broders' series of 537 patients, have no killing power, and, therefore, should be given conservative treatment, while those falling in grade four have a killing power of 100 per cent, and therefore it is useless to subject these unfortunate patients to a major operation.

On account of the nature of the original growth, it presenting an exposed ulcerating surface, more or less infection is present in a large percentage of the cases. Although this infection is usually mild, it frequently gives rise to inflammatory enlargement of the lymph glands draining the involved area. By palpatory examination it is often impossible to differentiate between this inflammatory enlargement and glandular metastasis. For this reason it is often necessary to start a glandular dissection and await the pathologist's report before deciding how extensive an operation to perform.

Since we are now able to grade these growths according to their degree of malignancy, it is possible to formulate certain rules for our guidance of their treatment. In the future we will be able to compare results from an intelligent, definite viewpoint and useless radical operations should be few and fatal conservative treatment rare.

Grade 1 (comprises 15.82 per cent). Complete removal of the cancerous area. Depending upon the size and location of the growth this may be best accomplished with the least disfigurement by the X-ray or radium in one case, a V excision in another, or a plastic operation in a third case.

Grade 2 and Grade 3 (comprising 83.05 per cent). A. When no glandular metastasis is present: Complete removal of the cancerous area together with a complete removal of all gland-bearing tissue from the submaxillary and submental triangles of both sides of the neck.

B. When glandular metastasis is present: 1. When the cervical glands are not involved

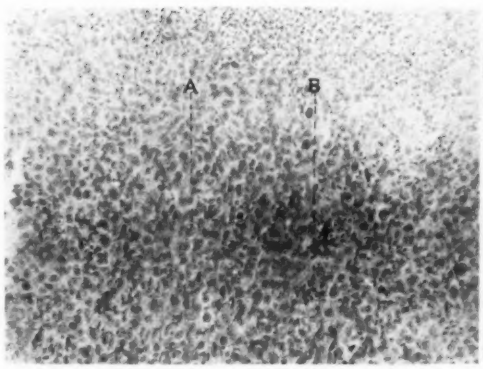


Fig. 4 (at left). Squamous-cell epithelioma of the lip, grade 4. *a*, Mitotic figure. *b*, "One-eyed cells." No differentiation present.

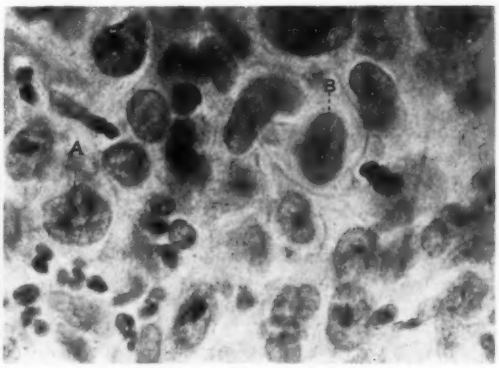


Fig. 5. Squamous-cell epithelioma of the lip, grade 4. *a*, Mitotic figure. *b*, "one-eyed cell." High power.

and only one group of the submental and submaxillary glands is involved: Complete removal of the cancerous area together with the removal of all gland-bearing tissue from both submental and submaxillary triangles and the removal of the upper superficial and deep cervical glands of that side of the neck corresponding to the metastatic growth.

2. When two or more groups of glands or when the cervical glands are involved: Complete removal of the cancerous area together with a complete block dissection of that side of the neck corresponding to the involved area, sacrificing the sternomastoid and omohyoid muscles and the spinal accessary nerve, and a dissection of the submental and submaxillary triangles of the opposite side.

Although such radical surgery will often prolong life a five-year cure is not to be expected in these cases.

Grade 4 (*comprising 1.11 per cent*). Palliative treatment. The classification of squamous-cell epithelioma into the four groups representating the various grades or degrees of malignancy is accomplished from the study of microscopic sections of the growth. It is based primarily on the degree of differentiation present although the presence of mitotic figures and "one-eyed cells," i.e. cells with large deep staining nucleoli, is to be taken into consideration. On account of the characteristics of adult squamous-cell tissue, differentiation toward the adult type is more easily recognized than in other kinds of tissue. While it is quite probable that other forms of cancer possess definite cellular characteristics corresponding to their degree of malignancy such characteristics have not been established.

If a squamous-cell epithelioma shows three-fourths of its tissue to be in a high state of differentiation with no mitotic figures and no "one-eyed cells" present, it is classed as Grade 1.

When approximately one-half of the tissue is differentiated and only an occasional mitotic figure and few "one-eyed cells" are present, it is classed as Grade 2.

When only one-fourth of the tissue is differentiated and mitotic figures are seen in every second or third field and two or more "one-eyed cells" are present in practically every field, it is classed as Grade 3.

When no differentiation is present and mitotic figures and "one-eyed cells" are freely distributed throughout the specimen, it is classed as Grade 4.

While it is easy to distinguish a Grade 1 from a Grade 4, it requires considerable experience to classify properly many of the growths which are on the border line between any two grades. However, it is surprising how seldom disagreement occurs between two persons familiar with the work and the frequency of squamous-cell epithelioma affords ample opportunity for any pathologist to familiarize himself with it.

CONCLUSIONS

1. Squamous-cell epitheliomata vary in their degree of malignancy.

2. On account of this variation in malignancy "cancer cures" do cure a certain percentage of lip cancers.

3. It is practical to grade squamous-cell epithelioma according to their degree of malignancy.

4. Rational treatment of squamous-cell epithelioma depends upon a proper grading as to their degree of malignancy.

CHOLECYSTOSTOMY OR CHOLECYSTECTOMY—WHICH?[1]

By MILES F. PORTER, M.D., F.A.C.S., Fort Wayne, Indiana

IS the question "Cholecystostomy or cholecystectomy—which?" a pertinent one? Has the gall bladder a function or functions to perform that are of importance to the economy or is it a practically useless and more or less troublesome appendage to be treated by the surgeon as is the vermiform appendix? Upon the answer to this question should depend in large measure the surgeon's treatment of the gall bladder. In a former paper (1) the writer presented evidence which seemed to establish the fact that the gall bladder in man was an important though not vital organ.

Rous and McMaster (2) in experiments on animals proved that the "concentrating activity" of the gall bladder was a function indicating that it was an organ of "special importance to the organism."

Robson (3) recently emphasizes the importance of the reservoir action of the gall bladder and says that "sufficient time has not yet elapsed to show whether the liver is injuriously affected by this pressure (after cholecystectomy) and absorption. . . . Whether cirrhosis of the liver and pancreas will result in a certain number of cases remains to be seen."

Rous and Larimore (4) found that both monolobular and diffuse intralobular cirrhosis were produced by ligation of the common duct.

Flexner's experiments prove that when pure bile is injected into the pancreatic ducts it produces pancreatitis, but when the bile is mixed with mucus before injection it is harmless.

R. C. Fravel (5) reports a series of cases of cholecystitis with and without stones, in a large percentage of which there was hypochlorhydria. He says "whether the gall bladder has a function or not the effect of its removal must be decided before we can dogmatically say that all diseased gall bladders should be removed." This author also refers to Holweg's (6) observation of 42 cholecystectomized patients suffering from gastric symptoms, in 83.3 per cent of whom hydrochloric acid deficiency was found, and his statement that a similar phenomenon followed cholecystectomy in dogs. He suggests that the acid produced in the stomach may be an index of the function of the gall bladder and advises against the removal of the gall bladder in those cases in which, from the acid content of the stomach, it seems capable of functioning.

It is contended by some that the value of a functioning gall bladder to the economy is not great enough to warrant the leaving of those even possibly dangerous. Says one: The "recurrence of gastric symptoms after cholecystostomy usually means that the gall bladder should have been removed." My own opinion and experience is not in accord with this. On the contrary my experience has been that continuation of symptoms after cholecystostomy means in the majority of instances either (a) overlooked or new-formed stones, (b) cholangeitis, (c) ulcer of the stomach or duodenum, or (d) pancreatitis. Now and again one finds in the literature a case wherein symptoms attributed to gall-bladder disease were found to be the result of a diseased appendix; however, the more frequent error consists in accusing the appendix when the gall bladder is the real culprit. The deductions made by many of those who champion the cause of cholecystectomy as against cholecystostomy are frequently fallacious. One finds many cases reported purporting to prove that cholecystectomy frequently relieves cases wherein cholecystostomy fails. In the majority of these reports a careful analysis of the individual cases will show that when the cholecystectomy was done a stone or stones were also removed. Jacobson (7) in his paper on the gall-bladder work done at the Peter Bent Brigham reports five secondary operations for overlooked stones. In four of these cases the primary operation was a cholecystectomy. In Jacobson's series (397) of cases "a normal gall bladder was removed in sixteen." Five of seven of these patients who were traced reported that they were "unimproved or in worse condition than before the operation." This report of Jacobson's illustrates the dangers of an overweening faith in cholecystectomy consequent upon an unwarranted suspicion of the gall bladder as a power for evil and an equally unwarranted belief in its uselessness. In a clinic manned by men believing that the gall bladder is an important organ, the bellies of these patients would have been closed perhaps after providing for drainage of the gall bladder, and the search for the real trouble continued at once on development of the fact that neither the gall bladder nor bile showed any pathology. This report seems to me also rather to prove the danger of routine operations in general and to emphasize

[1] Read at the meeting of the Western Surgical Association, St. Louis, December 9, 1921.

the force of Eastman's statement (8) that "it is mischievous to take out the gall bladder simply because it is an attractive operative procedure. If we had to deal with an infection of the gall bladder alone, this would be a very easy question to solve; but I have never seen a badly infected gall bladder that was not associated with infection of the ducts, or with infection of the pancreas. I believe we shall decide more and more to utilize the gall bladder for drainage in such cases" than to lead to the conclusion the writer reaches, viz., that "cholecystectomy is the operation of choice in cholelithiasis and cholecystitis whenever feasible." Evidence is rapidly accumulating which seems to show that infection of the gall tracts usually commences in the liver (9). This evidence minimizes the force of the argument that removal of the gall bladder is an important factor in the cure of cholangeitis and its results, and emphasizes the value of drainage. I am firmly convinced in my own mind that the cures which have followed cholecystectomy in cases wherein cholecystostomy failed, except those wherein additional stones were found, are to be explained many times on the basis of the drainage consequent upon the cholecystectomy with its resultant dilatation of the common duct and paralysis of the sphincter of Oddi, and not to the fact that a nidus of infection (i.e. the gall bladder) was removed, or to the fact that the "only serious contractile force in the biliary system has been cut out," as held by Archibald (10). Another argument advanced in favor of removal of the gall bladder is that a diseased gall bladder after drainage probably does not regain its power to function normally. This argument is refuted by the fact that numerous gall bladders have been examined after cholecystostomy and found normal, while there are recorded many hundreds of patients entirely free from gall-bladder symptoms following this operation. Personally I never knew an acute cholecystitis that failed of a cure from cholecystostomy, unless a stone had been overlooked, save one. This exceptional case was one of acute cholecystitis and cholangeitis with jaundice. The drainage was not kept up as long as it should have been, soon after it stopped there was an acute return of symptoms including jaundice, a second operation was refused, death ensued and at autopsy the common and hepatic ducts were found to contain pus and masses of grayish inspissated mucus.

W. J. Mayo (11) says: "A seriously infected gall bladder cannot exist without evidence of infection in the glands which drain it," and in such cases as fail to show the ordinary signs of infection "the mucosa will if examined show unmistakable evidence of chronic infection."

Now and again a typical gall-bladder stone will be the only physical finding lending color to the clinical picture of chronic cholecystitis. Yet again even this positive evidence may be absent and we have left only the appearance of the mucosa and the divided walls of the gall bladder to depend upon. Supposing all these signs fail, shall we remove a gall bladder on the clinical symptoms alone? It is not denied that a gall bladder may be infected to a degree requiring its removal and yet there be no signs of such infection that can be detected by the unaided senses, but I have never had such a case demonstrated to me. Until I have I shall continue to drain such gall bladders rather than remove them.

SUMMARY

Routine removal of the gall bladder is based upon the following assumptions:

1. That the gall bladder is an unimportant organ and that its removal is followed by no untoward results.

2. That all gall stones have their origin in a "primary cholecystitis" (12) and that recurrence or continuation of symptoms following cholecystostomy is usually due to the fact that the gall bladder was not removed. The facts are:

1. All evidence at hand supports the contention that the gall bladder is an important organ and evidence is not yet at hand to warrant the conclusion that its removal leads to no serious consequences.

2. Cholecystic disease including gall stones frequently if not usually originates in the liver. Routine cholecystectomy frequently fails to cure and leads to the removal of healthy gall bladders in over 4 per cent of cases.

CONCLUSION

The gall bladder should not be removed unless it has become useless or dangerous by disease.

REFERENCES

1. J. Am. M. Ass., 1917, lxix, 518.
2. J. Exper. Med., 1921, xxiv, 47. Abstracted Internat. Abst. Surg., 1920, xxxi, 453; 1921, xxxiii, 405.
3. Oxford Surgery, 1919, iii, 293.
4. J. Exper. Med., 1920. Abstracted.
5. Am. J. M. Sc., 1920, clix, 512.
6. Progressive Med., 1913, December.
7. Arch. Surg., 1920, i, 310.
8. J. Am. M. Ass., 1917, lxix, 1469.
9. MacCarty, Jackson. Collected Papers Mayo Clin., xii.
10. Ann. Surg., 1921, lxxiv, 432.
11. Keen's Surgery, viii, 452.
12. McGuire. Surg., Gynec. & Obst., 1920, xxxi, 617.

EDITORIALS

SURGERY, GYNECOLOGY AND OBSTETRICS

FRANKLIN H. MARTIN, M.D. ..... Managing Editor
ALLEN B. KANAVEL, M.D. ..... Associate Editor

JULY 1922

ON THE USE OF TRANSDERMIC APPLIANCES IN THE TREATMENT OF FRACTURES

WHETHER right or wrong, when an idea has once gained credence among surgeons it has a tendency to stick. This conservatism is good in so far as it prevents the too easy acceptance of half-baked theories; but it is unfortunate when it inhibits the introduction of new and valuable procedures, as it sometimes does.

For instance, our notions regarding the vulnerability of the peritoneum kept us for a long time out of the abdominal cavity, and our conviction that it was dangerous to irrigate the surface of the pleura prevented us from employing antiseptic solutions in the treatment of empyema.

Similarly, the fear of infection in fractures of the long bones has caused us to shy at the adoption of various useful methods of fixation, traction, and extension, which have been presented from time to time, such as the external bone-clamp, the wire-and-nail traction of Steinmann, the ice-tongs of Ransohoff, and the fixation of fragments by means of nails and screws inserted through the skin.

But in the face of this deeply rooted prejudice, an extensive experience in the Great War, added to that of civil practice, as reported by many observers, has demonstrated that when properly carried out the likelihood of infection in these procedures is slight and our fears have little justification.

In fact, from the evidence now at hand, it seems fair to assume that if the skin is well prepared there may be introduced through it into the bone, and left there almost indefinitely, screws, nails, wires, or ice-tongs, as indicated, with almost no danger of infection, provided these things are clean and that they fit the openings snugly without undue pressure or traction upon the surrounding integument, safety being doubly assured by means of a dry dressing and the occasional application of weak tincture of iodine.

When this principle is widely recognized, as it should be, it will open the way for a more simple and effective treatment of certain fractures in which the results leave much to be desired at the present time. Few surgeons will venture to deny, for instance, that traction by means of tongs or wires applied directly to the bone may be more effective and practicable than that obtained by strips of adhesive plaster attached to the skin; and, in addition to conserving motion of the adjacent joints, the method may be employed where the condition of the skin prevents the use of splints or adhesive. Likewise, the external bone clamp, the principles of which have been so well described by Parkhill, offers many advantages in the way of fixation and extension, in fractures of the more superficial

long bones, if only we were not afraid to use it. For instance,—the ease of application and removal (it sometimes may be inserted through mere punctures in the skin); the safety of placing the screws far from the line of fracture; the fact that the instrument is mostly external and not internal, like a Lane plate; and the firmness with which the fragments are held, because of the stability of the clamp and the size of the removable bone-screws.

From experience with the external bone clamp and traction by means of wires, nails, and tongs, the question of infection resolves itself into this: If proper cleanliness is observed, the danger is slight and distinctly less than in the use of an internal plate; because a minimum of manipulation is required and the line of fracture is not encroached upon by a foreign body. But if the operation is not clean, suppuration will supervene, as in any other surgical procedure, although the results are apt to be less disastrous because of drainage afforded by the openings through the skin. In addition, the screws can be removed at any time, thus eliminating all disturbing material. Unfortunately for a correct understanding of the situation, it often is more agreeable to attribute a septic disaster to something inserted through the skin than to an error in technique, although the comparative frequency of infections in connection with other operative procedures, such as the use of the Lane plate, should assist us in avoiding such misconceptions.

The notion that a late infection is apt to creep into the bone along screws, nails, or wires is largely theoretical as has been demonstrated regarding the external clamp and the Steinmann traction; and if such an infection should occur it is almost invariably trivial, being localized by the formation of an enveloping granulating channel.

In conclusion, whatever may be one's opinion regarding the usefulness of appliances inserted through the skin into broken bones, one should at least, in the light of existing evidence, recognize their practicability and safety.

LEONARD FREEMAN.

ILLUSTRATED CASE REPORTS AS AN AID IN SURGICAL TEACHING

ILLUSTRATED case reports as an aid in surgical teaching were presented at a recent meeting at which there were representatives of a number of teaching institutions. Comments led to the belief that the method is not widely employed and that its value is not generally appreciated; yet a trial of 5 years has proved it to be of much practical value and worthy of the consideration of clinical teachers.

When a clinical demonstration on a given subject is planned, it is usually well nigh impossible to secure cases which illustrate the lesions that are to be discussed and it is often impossible even to secure desirable cases which demonstrate the ultimate results of treatment. The difficulty is especially great if the same cases are requested to return serially for demonstration to successive sections of students or even once a year for successive classes. As a result, students see only those cases which are momentarily accessible, and these are for the most part the limited number which are in the hospital. Consequently, students do not profit by the material which has passed through the hospital in the past. Such material always presents cases which could be employed advantageously for teaching were the records in proper form.

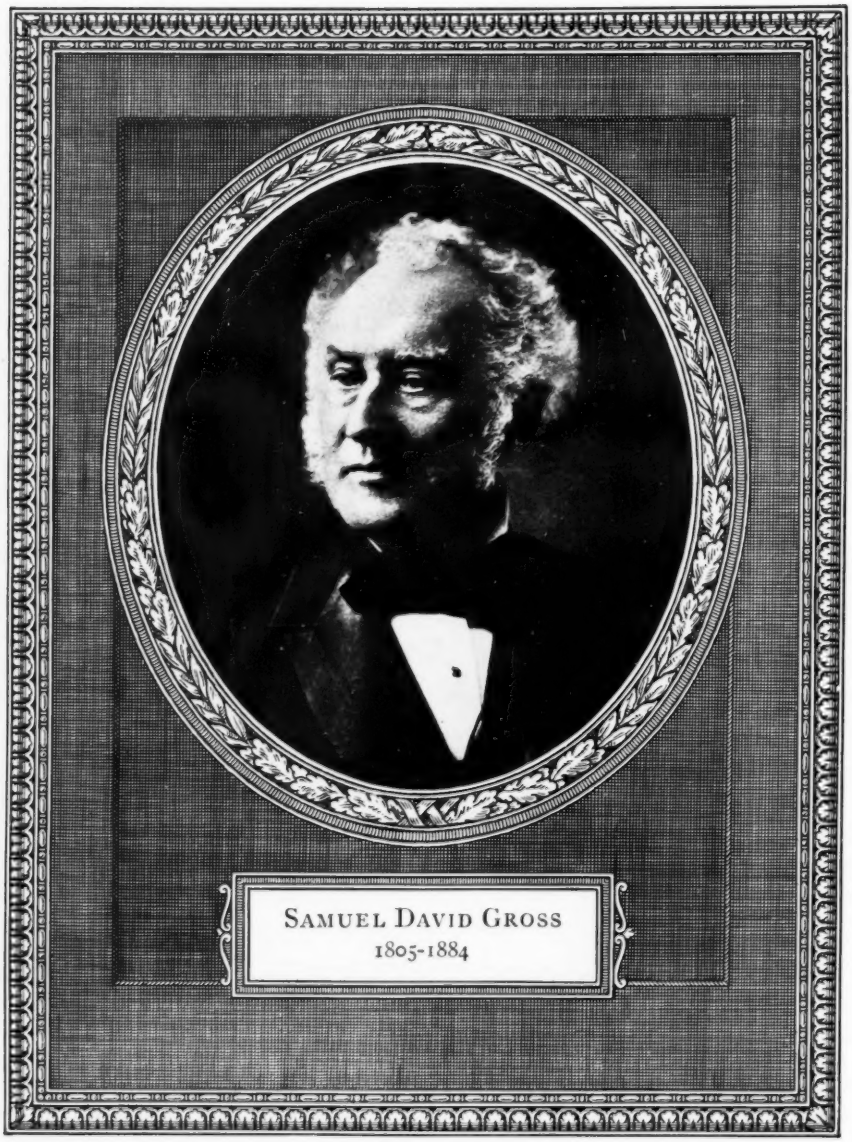
The deficiency of illustrative cases can be largely corrected by working up and filing type cases and unusual cases as they pass through the hospital, bearing in mind that

the hospital records are usually inappropriate and deficient for this purpose. Each member of the staff must be alive to the possible importance for teaching purposes of cases under his care. With this in mind the proper selection of cases for this purpose is readily made. There is prepared a careful summary of the history and physical examination of cases, ante-operative photographs and X-ray prints. After operation, photographs of the gross specimen removed and photomicrographs of typical areas are incorporated with the record. The postoperative course is summarized. Photographs after operation are included as well as the ultimate results as determined by the "follow-up" examinations. In this way a complete record of the case is available for demonstration to students. Duplicate cases need not be accumulated but type cases should be replaced as more illustrative ones are secured. With such material one can demonstrate in a convincing manner the full course of individual cases or groups of cases. If, in the course of a clinic, a postoperative case is demonstrated, one can often utilize to advantage the ante-operative records and photographs of similar cases.

The method lends itself best to lesions or diseases where some abnormality can be registered in photograph or X-ray plate, as, superficial tumors, skin lesions, exophthalmic goiter, osteomyelitis, myositis ossificans, Paget's disease, and other deformities. But the field is far more extensive than appears at first sight if pathological material is included. Of course photographs are not as perfect records as colored illustrations or models, but are fairly good substitutes. The case records are grouped under subjects. These may be arranged in albums with removable leaves, an album being devoted to each subject, thus enabling an instructor to secure very quickly material to illustrate a clinical demonstration.

In a few years there is accumulated complete records of groups of cases and unusual cases. The method requires some work and foresight but the results will amply repay the effort.

EUGENE H. POOL.



SAMUEL DAVID GROSS
1805-1884

MASTER SURGEONS OF AMERICA

FOREWORD

American surgery is cosmopolitan. Students from the United States have pursued their studies in all the great hospitals and universities of the world, each bringing home something of value which has been promptly introduced into American practice. The zeal and enthusiasm of the American student both at home and abroad has led to profitable scientific research and medical and surgical investigations, the value of which has been recognized internationally. Science has no country, but scientific men are more or less influenced by the conditions in their own countries, and each country has its heroic dead whose life work contributed to scientific advance.

Sir Berkeley Moynihan has pointed out the value to the medical student of biographies of British surgeons that are appearing in the *British Journal of Surgery*, and suggests that the biographies of great American surgeons of the past be written that their lives may be familiar to all, and their deeds an inspiration to surgeons of this generation. The Board of Editors of the American College of Surgeons has acted on this suggestion and will publish in each issue of SURGERY, GYNECOLOGY AND OBSTETRICS a biography with a portrait of an American surgeon who, before passing beyond, has done his work well.

WILLIAM J. MAYO.

SAMUEL DAVID GROSS

SAMUEL DAVID GROSS, the elder Gross, as we all called him in order to distinguish him from his brilliant son and successor, Samuel W. Gross, my old master whom I loved and admired with all the enthusiasm of boyhood more than 30 years ago, was born in Easton, Pennsylvania, in 1805.

His great-grandparents came from the Lower or Rhenish Palatinate in one of the emigrations of the Seventeenth Century, from that portion of Germany which, before its devastating wars, had been a veritable garden spot. The soldiers of Louis XIV had interpreted literally the order of the Sun King to Louvois to burn up the country. They killed three-fourths of the people and destroyed

four-fifths of the property. In the Palatinate, Wallenstein and Gustavus Adolphus, Tilly and Spinola, Condé and Turenne performed some of their most sanguinary actions. They left the Palatinate a gory wilderness with the blackened ruins of homesteads, buildings, and churches and the land harried by great flocks of ravening wolves.

In the veins of Gross ran the blood of the calm, scholarly, patriotic, broad-minded, deep-thinking, home-loving, and philanthropic Palatines; of those men who abandoned home and country for the rights of free speech, of liberty, and of conscience.

Gross was brought up on a farm. He loved the country all his life and he knew and appreciated its magic. Nature was to him an endless pleasure. Birds, animals, the woods and especially flowers were to him a source of the utmost delight. He loved the sunrise with its glory of color and the sunset with its splendor of flame. He loved the blue depths of the heavens, the sea, the majesty of the storm, the starlight, and the stream.

He knew familiarly the flora of this entire region of Pennsylvania. He always kept flowers in his office and about his house, and he insisted on seeing them in hospital wards. Once in a hospital I saw him tenderly lift a flower, sadly comment upon how soon it would fade, saying: "It has but one Spring and we mortals go almost as quickly for we have but a few Springs." He had within him something of the Wordsworth feeling: "It is my faith that every flower enjoys the air it breathes."

He was educated in the common schools and started to study medicine under Doctor Swift, of Easton. After he had begun his study in Doctor Swift's office he made up his mind that he had not sufficient education for the proper study of his profession and he went back to school, finishing in the famous Academy at Lawrenceville, New Jersey. He then renewed the study of medicine, entering the Jefferson Medical College of Philadelphia, and being an office student of the celebrated Dr. George McClellan.

On his graduation he started practice in Philadelphia and eked out a livelihood by hack literary work and translations. He spoke German and he acquired a knowledge of French in the course of a few months in order to translate Alphonse Tavernier's *Elements of Operative Surgery*, in 1829. He translated the *Manual of General Anatomy*, by Bayle and Hollard, in 1828, and Jules Hatin's *Obstetrics* in the same year. In 1829 he translated Johann Valentin von Hildenbrand's *Treatise on the Nature, Cause, and Treatment of Contagious Typhus*. Tavernier's was the first treatise on operative surgery published in America and attained great success.

In 1830, two and a half years after his graduation, he wrote a work on *Diseases and Injuries of the Bones and Joints*, and although the book became popular, the liberal publishers never paid him a cent. Want of practice in

Philadelphia obliged him to move back to Easton. After remaining there for a time, he went to Cincinnati and from there to Louisville, where his fame was made.

In 1839 he published his great treatise on *Pathological Anatomy*, the first in the English language. He received not a single cent for the first edition. The work was a great success and made him broadly known in this country and in Europe. The book was a popular textbook for more than a quarter of a century.

In 1851 he published a treatise on *Diseases of the Urinary Organs*. Up to this time there had been no satisfactory treatise in the English language, the only two existing being Brodie's *Lectures on the Diseases of the Urinary Organs* and William Coulson *On Diseases of the Bladder and Prostate Gland*, both published in 1842. This book, edited by the younger Gross, was still a popular textbook at the end of the 80's in the last century.

In 1854 he published his noted treatise on *Foreign Bodies in the Air Passages*. This was the very first attempt that had been made to present the subject in systematized form.

In 1859 he issued the first edition of his magnificent textbook on surgery; the greatest of his day, the first from America and probably the greatest ever written by one man. Recall the comprehensive nature of the title: *A System of Surgery, Pathological, Diagnostic, Therapeutic, and Operative*. The sixth edition was issued in 1882.

In the beginning of the Civil War, he wrote a *Manual of Military Surgery*. In 1861, he edited the *Lives of Eminent American Physicians and Surgeons of the Nineteenth Century*, writing several of the biographies himself. At times he edited medical journals. He contributed a multitude of articles to medical journals and had made some original researches of the first importance, especially those upon gunshot wounds of the intestines.

He had been a teacher in Cincinnati, in Louisville, in New York City before he came to his real home as a professor of surgery in the Jefferson Medical College. He was one of the greatest of surgeons and teachers. He founded the American Surgical Association. He was long the most influential member of the American Medical Association. He presided over the International Congress of Surgeons in 1876. He received high public honors in Germany, and was given the D.C.L. of Oxford, the LL.D. of Cambridge, the LL.D. of Edinburgh, and when actually on his deathbed, the LL.D. of the University of Pennsylvania.

His intellect was of the most comprehensive and luminous sort. He was a real surgical philosopher. Probably no greater mind was ever devoted to the science and art of surgery. He was a great man, a good man, and a gentleman; a master teacher and one who commanded not only the respect but the affection of the entire profession.

His old friend, Doctor Yandell of Louisville, wrote an epitaph which contains a just estimate of him. In this, Yandell states that Gross's life was one "unbroken process of laborious years," that he "filled chairs in four medical colleges in the Union and added lustre to them all." That "he recast surgical science, as taught in North America, formulated anew its principles, enlarged its domain, added to its art, and imparted fresh impetus to its study." Among the many books he composed is "*A System of Surgery* which is read in different tongues, wherever the Healing Art is practiced." "With a great intellect, carefully trained and balanced, he aimed with undivided zeal at the noble end of lessening human suffering and lengthening human life, and so rose to the highest position yet attained in Science by any of his countrymen."

The writer of this brief memoir beheld the mighty leader a great many times, heard him lecture frequently and watched him operate, and in him always saw the embodiment of surgical learning, dignity, and distinction, and felt that fifty years of American surgery were speaking through his lips.

J. CHALMERS DACOSTA.

TRANSACTIONS OF SOCIETIES

CHICAGO GYNECOLOGICAL SOCIETY

REGULAR MEETING HELD DECEMBER 16, 1921, DR. W. C. DANFORTH PRESIDING

THE INFLUENCE OF OVARIAN SECRETION ON HÆMORRHAGIC TENDENCY

DR. ARTHUR H. CURTIS: I very recently operated upon a young woman of about 30 or possibly a little older who had two large chocolate cysts of the ovary, one the size of a fist, the other four or five times as large. There was almost no ovarian tissue left in the cortex of these cysts. This patient had been under the care of Dr. Oliver S. Ormsby for over 7 years. Every time the cutaneous surfaces were bruised ever so slightly she developed a hæmorrhagic area, at times as large as a saucer, over various parts of the body surface.

There arises the question as to whether the disturbed secretion of the ovary is an essential factor in the development of the hæmorrhagic tendency. I suppose some of you have seen similar cases, and I wish to ask whether other members have encountered similar dyscrasias through disturbances of the ovaries. The giving of a hypodermic injection the morning of the day preceding operation gave a great deal of pain and on the morning of the operation was accompanied by an ecchymosis that covered the entire arm. No hæmorrhage occurred on removing the ovaries. The coagulation time was 7 minutes 20 seconds before operation and 7 minutes 40 seconds a few days ago.

She had had feelings and symptoms of the menopause and I was wondering whether it was the ovarian cysts that disturbed menstruation.

THE X-RAY IN PREGNANCY; X-RAY EXAMINATION OF TEETH DURING PREGNANCY

DR. CARL HENRY DAVIS: In view of the paper on the use of X-ray in pregnancy which is to be discussed tonight, I wish to show films of a twin pregnancy and a number of mouth films which show the importance of having all dead teeth X-rayed.

In the past 2 years it has been my unusual experience to have delivered six women of twins and one of triplets. In two instances it was impossible to determine the relative positions of the babies on account of excessive fluid and X-ray plates were secured. In the first case we were able by comparison of different films to demonstrate the babies' heads and determine the relative positions. This film of the second case was made with the Potter-Bucky diaphragm which makes it possible to get much clearer pictures in this type of X-ray work. It shows both babies clearly.

Since Talbot's paper on focal infections in the etiology of toxæmias, I have been much more careful than formerly to have the teeth examined frequently during pregnancy. But dentists frequently maintain that there is no need to X-ray dead teeth and tell the patient that the mouth is in good condition. Within a year six patients have developed a more or less severe toxæmia which I believe might have been avoided had infected teeth been removed early in pregnancy. You will note in the films which are being passed that practically all of the dead teeth show infection.

It is my belief that the physician should require X-ray examination of all dead teeth and have any showing definite infection removed as early as possible in pregnancy.

ADENOMA OF THE UTERUS; REMOVAL OF FOREIGN BODY FROM THE BLADDER

DR. W. C. DANFORTH: Most of you have read the paper by Dr. Sampson on "Adenoma of the Uterus." In this woman a mass existed which was definitely palpable in the cul-de-sac with diminished mobility and quite tender. Operation disclosed hæmorrhagic cysts of the ovaries, the larger one being on the left side and containing thick chocolate-colored fluid. A complete hysterectomy was done, there being very dense adhesions between the posterior wall of the uterus and the anterior rectal wall which rendered operation a rather difficult one. Upon section, an adenomatous growth was found in the posterior uterine wall. We found no endometrium or endometrium-like epithelium in the ovaries in this particular instance.

About 2 years ago Dr. Barrett showed us a very useful little stunt for removing foreign bodies from the bladder. He suggested the use of a small Nitze water cystoscope alongside of which through the urethra might be passed a long, narrow Kelley vesical forceps. I had no occasion to make use of this bit of technique until a few days ago, when a woman came into the hospital with a piece of catheter in the bladder. By introducing the cystoscope and Kelley forceps, it was possible with very little

difficulty to grasp the catheter and, after removing the cystoscope, to draw out the piece of catheter. It seems to be a rather valuable bit of technique.

THE TEST OF LABOR

DR. GEORGE LEE presented a paper entitled "The Test of Labor." (See p. 63.)

DISCUSSION

DR. RUDOLPH W. HOLMES: It is refreshing to have Dr. Lee present a summary of his obstetrical work, because of late years we have heard much of operations on the pregnant, and little of pure obstetrical care in labor.

In regard to one point I cannot quite agree with Dr. Lee on the question of bony obstruction. The crux of the problem is the cephalopelvic relationship. If there be a disproportion, it is an indication that perhaps there may be an insurmountable difficulty, but it does not necessarily follow. The molding of the head and the resistance of the soft parts play an important part. Take, for example, a vera of 9 centimeters, or as some would have it, 8 or 8½ centimeters, as the index between the relatively and absolutely contracted pelvis, spontaneous labor may be reasonably expected in a fully 90 per cent of such cases, and will terminate spontaneously, or at most in a not difficult forceps operation. In women with the above limitations, it has been my fixed rule that no valid determination of the woman's potential possibilities may be fixed unless she has had a real test of labor. I examined a woman recently who had a conjugata vera of 6 centimeters, yet she stated she had given birth in a short labor to a baby weighing 6 pounds (Swedish). I would have had some five to ten times the number of cæsareans I have had if I had relied on mere mensuration, and not on the test of labor.

DR. CHARLES S. BACON: I would like to ask Dr. Lee what he means by fœtal exhaustion. How is that determined?

I would question the indication for high forceps of posterior right or left position at the inlet. I am somewhat doubtful whether that has any bearing, for if descent occurs rotations forward will take place. The important thing is the treatment of these cases of delay at the inlet. I would not always consider a previous labor of 15 or 20, or even 30 hours as a complete "test of labor." The real "test" must be managed properly. With proper support of the patient and the relief of pain after 40 to 50 hours, the head may descend so that a low forceps operation can be made. Generally in these cases delay is due to lack of dilatation of the cervix and much time may be required to overcome this obstacle.

DR. CARL HENRY DAVIS: Dr. Lee's paper brings up the question of just how we should manage the cases where the head is high, particularly with the occiput posterior, when complicated by premature rupture of the membranes. It has sometimes seemed that the life of the baby might have been conserved in this type of case had we promptly introduced a Voorhees bag to dilate the cervix instead of leaving that entirely to the fœtal head. I would like to have Dr. Lee tell us his attitude relative to the use of a bag in this type of case.

DR. MARK T. GOLDSTINE: I would like to know how Dr. Lee picked this one hundred consecutive cases and how they were done, because the hundred shows a good deal more abnormality than is usual in the average one hundred cases. It is unusual to have only 15 spontaneous deliveries in primipara out of 51. Thirty-nine multiparæ in this series is a high percentage to require help. To my mind he could not have picked a harder one hundred cases. I venture to say that if he chooses another hundred his percentage in favor of the test of labor will show up far better than this hundred showed.

DR. W. GEORGE LEE (closing the discussion): Dr. Holmes I think misunderstood me in referring to bony obstruction. My point was that where the bony obstruction is great enough to influence one to do a cæsarean section primarily or after a few hours, in the first stage of labor, no test of labor is given, and hence an abdominal section is better done without waiting for the few hours of undetermined labor.

In answer to Dr. Bacon's question concerning fœtal exhaustion, I used that as a term for marked alteration in the fœtal heart tones, showing changes, and in some instances the passage of meconium. The change in fœtal heart tones is the most important sign.

In the matter of posterior fixation I think Dr. Bacon rather misunderstood me. I did not recite that as a factor for interference but merely that as a complicating factor this was present in an unusual number of cases.

As to the dry labors, I think the bags are beneficial in helping to dilate the cervical portion, but I think in many cases the constriction ring is on the head, and the bag does not help. I think in these cases the longer we delay the more firmly set is the constriction ring and an earlier interference seems justified. We then aid in dilating by aiding the muscle to overcome the resistance before the lower segments have "dug themselves in," so to speak.

Speaking of this particular series, Dr. Goldstine, I presented them because they seemed to me a very unusual series of cases, but I should dislike to think I would get another hundred with as great a number of cases needing interference. The majority of these cases I saw throughout labor.

ROENTGENOGRAPHY IN OBSTETRICS

DR. D. A. HORNER presented an inaugural thesis entitled: "Radiography in Obstetrics." (See p. 67.)

DISCUSSION

DR. JOSEPH B. DELEE: This work has been done under my eye for the last 2 years, and I feel that we are achieving some real results. I formerly looked

upon the measurement of the pelvis as an almost impractical thing, but it seems to me now we have a scientific method of doing it. With the modern treatment of contracted pelvis we do not need these refined methods, but sometimes we are glad to have them. For example, in the breech presentation where it is highly desirable to give the people a live baby and we cannot give the test of labor. I shall refer to the test of labor only so far as the pelvic contraction is concerned. To give the woman the test of labor is merely a subjective process. I do not want such a test that proves her absolute physical inability to force a child through the pelvis. What I consider a good test of labor is when a woman has demonstrated to me her inability to complete the labor herself, with safety to both concerned. Some damage has been done to the woman's nervous system, to the soft parts and possibly to the baby if the absolute test has been given. With this new X-ray method I think we shall escape the necessity of giving these women the prolonged test of labor.

There are many interesting things in the plates shown. The pictures of the dead baby with macerated bones is a real advance and should be very useful. It is not always possible to tell beforehand whether the fœtus is dead, and since this may eventuate in infection, the X-ray may save an endless amount of trouble.

So far as the use of the X-ray on the fœtus is concerned, I think there is no danger. The demonstration that it is possible to measure the pelvis and also the baby is the most important thing in the paper, and it puts the real measurements of the pelvis more firmly on a scientific basis than ever before.

As Dr. Horner stated, there is a requirement in some states that there be a physical examination prior to marriage, and I believe it would be a very good thing for women to know if they are anatomically fit to have children, and this method should be helpful there.

Dr. Edward L. Cornell: Dr. Horner failed to mention the diagnosis of ectopic pregnancy by means of X-ray as demonstrated by me in a case where we employed pneumoperitoneum on a rather obscure mass in one of the tubes. The patient had gone by for a period of about 6 weeks. The only symptom was a complaint of pain. We did a pneumoperitoneum, demonstrated the mass in the tube, and removed the ectopic before rupture.

Dr. Irving F. Stein: At Michael Reese Hospital we have been much interested in similar endeavors and I would like to show a picture of one case in which life had not yet been felt. The patient, a para-II, age 19, presented herself at the prenatal clinic having had one normal labor, and said that about one month previously the water had escaped and that she felt no life. We were unable to demonstrate either fœtal movement or heart tones in the clinic, or the ward where we watched her for 6 days. The X-ray film showed the head very distinctly and the fœtal spine in this area, the fœtus lying transversely with the back presenting. This did not show with any great degree of clearness on account of the density of the bones at this age. The question of the age of this fœtus was somewhat difficult to determine because the fœtus when it was expelled, about 10 days after the picture was taken, was macerated and putrid, was expelled during the night, and was not saved for measurement. The fundus on examination was in anteflexion and filled the whole true pelvis, and the os was open to the extent of the finger-tip. This patient, who had already had one baby, denied that she had felt life. Her last period was on October 28, 1920, and she presented herself at the clinic on April 18, 1921, so she then should have been about 5½ months pregnant. The water had escaped a month before, so the fœtus probably developed to 4½ months. We made daily examinations to determine whether life was audible through the stethoscope, but were unable to get either heart tones or other evidence of life. I think this case is decidedly younger than the one Dr. Horner showed.

Dr. D. A. Horner: This work has been done in Dr. DeLee's laboratory and I wish to thank him for the opportunity of being able to do it. I think that with a little more application of time to the problem of measurement of the pelvis, something really simple and valuable will be developed; something that will be accepted as essential. This method is not original with me. I am simply carrying out experiments in accordance with the method of Chamberlain and Newell.

To illustrate the case referred to by Dr. Cornell, I present this plate. It shows the pneumoperitoneum, the uterus slightly enlarged and off to one side and the tumor mass distinctly visible, with an enlargement of the tube and a portion of the ovary on the other side. The diagnosis of early pregnancy and tumor was made, I think, before the picture was taken. From the history of amenorrhœa and the conditions revealed in this plate, a diagnosis of extra-uterine pregnancy was made. Operation confirmed the diagnosis.

Dr. N. Sproat Heaney: Dr. Horner spoke about the plate of the woman who was 5 months pregnant and said this woman was 5 months pregnant according to the menstrual period and 5 months according to the plate of the fœtus. He did not say how he determined the age of the fœtus by a plate. Perhaps he did not wish to be so accurate as this although he made a very definite statement regarding this. I am not aware that one can tell accurately regarding the age of a fœtus but that we only receive relative information from the size of the bones and their density even when these factors are determinable.

Dr. D. A. Horner: My remarks pertaining to roentgenography of the unmarried woman to determine her fitness to marry and bear children, hold true for all women giving histories of previous obstetrical difficulties. There are numerous communities in which there is no one able to take care

of the borderline cases correctly. Many large sized towns exist in which obstetrics is of the most inferior sort, and in these as in all localities occur cases like the one I presented, where there was an overriding of an inch over the pelvic brim. Nature is depended on. Difficulty is discovered later. Then comes faulty interference. Not a few die as a result. One capable of deciding early and performing the different types of cæsarean section, if at hand, would eliminate this question of fitness to marry as judged from the obstetrical viewpoint.

As to the 5 months' fœtus, all of these pictures are taken at a standard height. The ray comes from the highest altitude which the stand permits. Therefore, every picture we take can be graded; a standard for every period can be set for that particular distance. At a distance of six feet, if we were taking them from that distance, they would be almost the same size as the fœtus *in utero*. The works of Hess and Adair, published not so long ago, gives us ossification details to obtain the age of the fœtus. Any textbook of obstetrics gives measurements which can be applied here. We judged that this was 5 months by comparing it with the film size of our 6 months' fœtus.

Dr. N. Sproat Heaney: I am not a radiologist nor a mathematician, and am, in consequence, not able to speak upon the technical points involved in Dr. Horner's paper. There are a few practical facts, however, which I do know. In reference to measuring the size of the 5 months' fœtus previously spoken of, if our constant factors are the heights of the tube and the level of the plate the object which we are taking is the variable point and if we are to measure its size we are to first determine its location. There is a considerable range of possible mobility in a 5 months' pregnancy. From the cervix to the fundus is a considerable distance, particularly since at this time we have a relative hydramnios. At this period of gestation the size of the uterus is disproportionate to the size of the fœtus. For that reason I am not convinced by the evidence that Dr. Horner has given us, that he can determine the size of the bones of a 5 months' fœtus. I, of course, may be mistaken in this and ask for further elucidation.

Dr. D. A. Horner: If you will recall, the unknown line is purposely put at an angle. It is immaterial how far it is from the plate as long as it is below the target. Its length is solved with those equations and figures (indicating) obtained with plane geometry. This may be a line on the fœtal head or on the pelvis, it will make no difference. The ultimate figure as shown in the model is a right trapezoid with the unknown line at the top. If you lift that line any distance above the plate or lower it, the equations remain identical.

Dr. Rudolph W. Holmes: Dr. Horner has presented a very interesting study, but I wonder what real clinical value his mathematical-geometrical deductions have. Of what value is the 76/100 unless you likewise may have the same precise mathematical figures for the whole pelvis from inlet to outlet, both anteroposterior as well as transverse. Further we must demand the same mathematical determination of the fœtal head in all obstetric planes. Again, no adequate determination of pelvocephalic relationship is realized unless the condition of the maternal soft parts and the degree of moldability is properly interpreted. As a theoretic consideration Dr. Horner's paper is a treat, as a practical suggestion for the bedside I fear it is not practicable. After all, the acumen of the trained obstetrician aided by an intelligent palpation offers more than any method of precise mensuration.

Dr. D. A. Horner: If we get the head through the pelvis we are satisfied that the rest of the fœtus will come through. The problem is not a matter of the soft parts. The greatest resistance we have to overcome is not the cervix or other soft structures, but the bony pelvis of the mother. Someone a while ago, advised using a bag in the difficult cases, stating that it would overcome the soft parts. Manual dilatation or incisions of the cervix and vagina will also overcome the resistance of these soft parts if the difficulties of the labor arise there. The resistance of other normal structures is negligible.

CORRESPONDENCE

THE BIOGRAPHY OF EPHRAIM McDOWELL

To the Editor: I am glad to have this opportunity to reply to Dr. L. S. McMurtry's letter published in correspondence in the May number of SURGERY, GYNECOLOGY AND OBSTETRICS concerning the biography of Ephraim McDowell.

As fully explained in the foreword, the biography is the outcome of a personal experience of mine in Berlin in 1911, when I was told that the belief was current in Germany that McDowell's first ovariotomy was performed upon a negress. In clearing up this doubt, I must repeat that I was impressed with the conflicting versions, dates, and the omissions that I encountered in print and correspondence. I have dealt with these at length in the biography.

My chief aim in writing the biography of Ephraim McDowell was to arouse the interest of the medical profession and of the public in this benefactor to untold coming generations, so that he might receive honors commensurate with the importance of his labors.

In trying to fulfill this ambition I was careful not to overlook any opportunity to emphasize as effectively as I knew how this long-continued neglect to do him justice. But I did not have the least thought of offending any person or organized body of persons. I relied on the breadth of view of my readers to appreciate the magnitude of this neglect in comparison with his work and to understand my earnestness in trying to secure just recognition for him among his own countrymen. I was also humbly endeavoring to bespeak for him attention beyond our shores. One who so profoundly influenced his profession and the human race for all time to come does not belong to any country. He belongs to the world, and his work was therefore not written entirely for America and the present period.

These aims determined not only the frankness but also the fullness with which the subject was handled. Every conflicting detail of any possible value, as well as the early historic atmosphere, was included with a view of making the presentation as clear as possible to Americans and foreigners living today and in future periods. I tried to do this, as one reviewer wrote, "before the lapse of time would make only tradition of these events."

Concerning our version of the removal of the remains of Dr. and Mrs. McDowell, I will say that it is similar to that published by his granddaughter in 1890, and so far as I have been able to learn it has until now remained unchallenged. Furthermore, this same version was corroborated and even elaborated at the first grave of Ephraim McDowell in the Shelby burial ground in 1912, by a kinsman of his in the presence of a witness. I might add that the topographical appearance of the former grave lent color to this kinsman's remarks, and the discovery of the fragments of the first tombstone followed later.

In my book I have also expressed the hope that other biographies will be written, and I have included the minute details with a thought of aiding others in just such an undertaking.

Dr. McMurtry's idea of a lectureship in the American College of Surgeons, similar to the Hunterian in the Royal College of Surgeons, has my unqualified support and approval. If the book had led to such a thought it was not written in vain.

AUGUST SCHACHNER, F.A.C.S.

Louisville, Ky.

THORACOSCOPY

To the Editor: In a paper on thoracoscopy by Dr. H. C. Jacobaeus published in the March number of SURGERY, GYNECOLOGY AND OBSTETRICS, I find a statement which is so at variance with the experience of thoracic surgeons in America that I can not let it pass unchallenged.

Dr. Jacobaeus says: "For, as we know, we have nothing for examining the chest cavity which corresponds to the exploratory laparotomy for examining the abdominal cavity."

Now, as a matter of frequent experience I have found, and I am sure that other thoracic surgeons have also found that a wide intercostal exploratory operation is far more thorough than the most complete exploratory laparotomy and this exploratory thoracotomy is practically devoid of shock, most of the patients being able to be out of bed 4 or 5 days afterward and, in my experience, some even earlier. Contrasted with the condition of a laparotomized patient with complete exploration of the abdominal cavity the picture is most convincing. Also, at such an operation specimens may be removed with the minimum danger and a more perfect understanding of all the patho-

logical conditions present can be had than in any other way.

I agree with Dr. Jacobaeus that the thoracoscope may have its uses, especially the one for which I understand it was originally devised, namely, to divide adhesions in tuberculous cases preparatory to lung collapsing pneumothorax, but thoracoscopy has shown in the doctor's 50 patients 10 cases, or 20 per cent, in which there were complications which must be considered serious, so the procedure can hardly be considered in the light of a minor surgical examination.

I shall watch with great interest the further development in this method of exploration of the chest cavity.

HOWARD LILIENTHAL.

New York.

INGUINAL HERNIOTOMY UNDER REGIONAL ANÆSTHESIA

A CORRECTION

In my article on "Inguinal Herniotomy Under Regional Anæsthesia," which appears on page 398 of the March, 1922, issue of SURGERY, GYNECOLOGY AND OBSTETRICS, an error has been made in the placing of the illustrations. Figure 8 should be transposed to appear over legend for Figure 5; and Figure 5 should be transposed to appear over Figure 8.

GASTON LABAT, M.D.

Paris, France.

BOOKS RECEIVED

Books received are acknowledged in this department, and such acknowledgment must be regarded as a sufficient return for the courtesy of the sender. Selections will be made for review in the interests of our readers and as space permits.

TUMOURS, INNOCENT AND MALIGNANT; THEIR CLINICAL CHARACTERS AND APPROPRIATE TREATMENT. By Sir John Bland-Sutton, LL.D., F.R.C.S. 7th ed. London, New York, Toronto, and Melbourne: Messrs. Cassell & Company, Ltd., 1922.

THE THYROID GLAND; CLINICS OF GEORGE W. CRILE AND ASSOCIATES. Philadelphia and London: W. B. Saunders Company, 1922.

SURGICAL AND MECHANICAL TREATMENT OF PERIPHERAL NERVES. By Byron Stookey, A.M., M.D. With a chapter on Nerve Degeneration and Regeneration by G. Carl Huber, M.D. Philadelphia and London: W. B. Saunders Company, 1922.

RADIUM THERAPY. By Dr. Frank Edward Simpson, A.B., M.D. St. Louis: C. V. Mosby Company, 1922.

THE PLACE OF VERSION IN OBSTETRICS. By Irving W. Potter, M.D., F.A.C.S. St. Louis: C. V. Mosby Company, 1922.

TRANSACTIONS OF THE TWENTY-SECOND ANNUAL MEETING OF THE AMERICAN PROCTOLOGIC SOCIETY. Boston, June 3, 4, 6, 1921. New Bedford, Massachusetts: George H. Reynolds, 1922.

TRANSACTIONS OF THE AMERICAN GYNECOLOGICAL SOCIETY. Vol. xlvi, 1921. Edited by George Gray Ward, Jr., M.D. New York, 1921.

TRANSACTIONS OF THE AMERICAN SURGICAL ASSOCIATION. Vol. xxxix. Edited by John H. Jopson, M.D. Philadelphia: Wm. J. Dornan, 1921.

THE SURGICAL TREATMENT OF NON-MALIGNANT AFFECTIONS OF THE STOMACH. By Charles Greene Cumston, M.D., and Georges Patry, M.D., with an introduction by Sir Berkeley G. A. Moynihan, K.C.M.G., C.B., M.S. Philadelphia: J. B. Lippincott Company, 1921.

LES OCCLUSIONS AIGUES ET SUBAIGUES DE L'INTESTIN. By A. C. Guillaume. Paris: Masson et Cie, 1922.

OPIATE ADDICTION; ITS HANDLING AND TREATMENT. By Edward Huntington Williams, M.D. New York: The Macmillan Company, 1922.

CHIRURGIE DE L'APPAREIL GÉNITAL DE LA FEMME. By R. Proust and J. Charrier. 5th ed. Paris: Masson et Cie, 1922.

THE PRINCIPLES OF PHYSICS AND BIOLOGY OF RADIATION THERAPY. By Dr. Bernhard Kroenig and Dr. Walter Friedrich. Authorized English edition with appendix. New York: Rebman Company, 1922.

MANUAL OF OBSTETRICS WITH CHAPTER ON ENDOCRINOLOGY. By John Osborn Polak, M.Sc., M.D., F.A.C.S. 2d. ed. New York: Physicians and Surgeons Book Company, 1922.

OBSTETRICAL NURSING; A TEXTBOOK ON THE NURSING CARE OF THE EXPECTANT MOTHER, THE WOMAN IN LABOR, THE YOUNG MOTHER AND HER BABY. By Carolyn Conant Van Blarcom, R.N. New York: The Macmillan Company, 1922.

TASCHENBUCH DER FRAUENHEILKUNDE. By Dr. Koblanck. 3rd ed. Berlin and Vienna: Urban and Schwarzenberg, 1922.

COLLECTED PAPERS OF THE MAYO CLINIC. By Mrs. M. H. Mellish. Vol. xiii, 1921. Philadelphia: W. B. Saunders Company 1921.

BIRTH STATISTICS FOR THE BIRTH REGISTRATION AREA OF THE UNITED STATES. 1920. Sixth Annual Report. Department of Commerce, Bureau of the Census. W. M. Stewart, Director. Washington: Government Printing Office.

CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

TWELFTH ANNUAL SESSION, BOSTON, OCTOBER 23–27, 1922

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PLANS FOR THE BOSTON MEETING

UNDER the guidance of a representative committee of Boston surgeons the plans for the twelfth annual session of the Clinical Congress of the American College of Surgeons, to be held in Boston, October 23–27, 1922, are well under way. In general the plans for the Boston meeting will conform to those of recent sessions of the Congress, and will include clinical demonstrations in the hospitals and medical schools during the morning and afternoon hours daily, with scientific sessions each evening.

Conferences on problems related to the hospital standardization program of the College are to be held Monday morning and afternoon in Jordan Hall. Papers will be contributed and discussed by Fellows of the College, hospital superintendents, trustees, nurses, and others interested in hospital problems.

At the Presidential Meeting, to be held in Symphony Hall on Monday evening, the President-Elect, Dr. Harvey Cushing, will be inaugurated and deliver the annual address. On the same evening Professor Raffaele Bastianelli of Rome, Italy, will deliver the John B. Murphy Oration in Surgery.

Clinical demonstrations at the hospitals and medical schools will occupy the morning and afternoon hours of Tuesday, Wednesday, Thursday, and Friday, and under the leadership of the Committee on Arrangements as noted above, we may expect a program of clinics and demonstrations to be developed that will accurately and completely represent the clinical activities of that great medical center. All departments of surgery will be represented in the clinical program, including gynecology, obstetrics, orthopedics, urology, surgery of the eye, ear, nose, throat and mouth, experimental surgery, surgical pathology, roentgenology, etc.

An interesting program of papers and discussions dealing with surgical subjects of present-day importance is being prepared by the Executive Committee of the Congress for sessions in Jordan Hall on Tuesday and Thursday evenings, in which eminent American and European surgeons have been invited to participate.

On Wednesday evening members of the Congress will be guests of the Boston Surgical Society at a special meeting in Jordan Hall for the presentation of the Bigelow medal.

The tenth convocation of the American College of Surgeons will take place on Friday evening in Symphony Hall, when fellowship in the College will be conferred upon a group of American and Canadian surgeons, and honorary fellowships upon the distinguished foreign guests.

General headquarters for the Congress will be established at the Copley-Plaza Hotel where the large ballroom, foyers adjacent thereto, and other large rooms on the main floor have been reserved for the use of the Congress, and will be utilized for registration and ticket bureaus, bulletin rooms, etc.

The annual business meeting of the American College of Surgeons and the Clinical Congress will be held in Jordan Hall on Thursday afternoon at 3 o'clock.

Application for reduced railway rates has been made to the railway passenger associations, and it is confidently expected that a substantial reduction in fares will be granted for the meeting.

LIMITED ATTENDANCE—ADVANCE REGISTRATION

It has been found necessary in recent years to adopt the plan of limiting attendance because of the popularity of these annual clinical meetings. Such plan necessitates registration in advance on the part of all who wish to attend. The limit of attendance will be based upon the result of a survey of the amphitheaters, lecture rooms, and laboratories in the several hospitals and medical schools as to their capacity for accommodating visitors. When the limit of attendance has been reached through advance registration no further applications can be accepted, hence the necessity for early registration. It has been our experience in recent years that the limit of attendance will be reached some weeks in advance of the meeting.

CLINIC TICKETS

The use of special clinic tickets has proven an efficient means of providing for the distribution of the visiting surgeons among the several clinics, and as well insuring against overcrowding. The number of tickets issued for any clinic is limited to the capacity of the room in which that clinic is to be given. Attendance at all clinics and demonstrations is controlled by means of such clinic tickets, which are issued each morning at 8 o'clock for that day's clinics.

As at previous meetings, a complete schedule of the day's clinics will be posted on bulletin boards at headquarters during the afternoon of the preceding day. After the program has been so posted, reservations for tickets for the next day's clinics may be filed, the tickets to be issued the following morning at 8 o'clock. A printed program will be issued each morning which will contain the complete clinical program for the day, with an announcement of evening sessions and other information.

REGISTRATION FEE

Plans for the Congress provide that no financial burden may be imposed upon the members of the profession in the city entertaining the Congress. Hence a registration fee of $5.00 is required of each surgeon attending the annual clinical meeting, such fees providing the funds from which to meet the expenses of conducting the meeting. To each surgeon on registering in advance is issued a formal receipt for the registration fee, which receipt is to be exchanged for a general admission card upon his registration at headquarters in Boston. This card, which is non-transferable, must be presented to secure special clinic tickets and for admission to the evening meetings.

STATE AND PROVINCIAL SECTIONAL MEETINGS OF THE CLINICAL CONGRESS OF AMERICAN COLLEGE OF SURGEONS

The Spring series of group meetings of the College came to a close with sessions of the New England Section at Portland, Maine, May 15 and 16, and of the Maritime Provinces at Halifax, Nova Scotia, May 19 and 20.

NEW ENGLAND SECTION

The Portland meeting was the largest medical gathering which the state of Maine has ever seen, and, due to an efficient local committee, all arrangements ran with perfect smoothness. An interesting and well balanced clinical program was arranged at the Maine General Hospital, the Eye and Ear Infirmary, and the Children's Hospital.

Over four hundred people, many of them laymen, crowded the hospital conference on Monday afternoon and listened to a program devoted to hospital betterment. Speakers from the central office included the director general of the College, Dr. Franklin H. Martin, Reverend C. B. Moulinier, S. J., president of the Catholic Hospital Association, and Mr. T. E. Allen.

In addition, Dr. John Osborn Polak presented the advantages of standardization to the surgeon; Dr. George H. Stone, superintendent of the Eastern Maine General Hospital, explained the hospital executive's attitude toward standardization; and Mr. Robert Jolly, superintendent of the Baptist Hospital, Houston, Texas, spoke on "Why the Public is Interested in Better Hospitals."

The round table discussion on hospital betterment, which proved such a great success in the South, concluded this program. Under the leadership of Dr. M. T. MacEachern, superintendent of the Vancouver General Hospital, and director general of the Victorian Order of Nurses, an instructive discussion of pertinent hospital problems was held with speakers from the audience participating. This round table discussion is steadily increasing in value and serves to give local hospital superintendents an opportunity to discuss their specific problems.

Portland citizens turned out *en masse* to the community health meeting. They filled the City Hall Auditorium and crowded the balcony. An audience of more than 2,500 people listened with the greatest interest and showed that the public of Portland possessed a community conscience in hospital affairs.

Dr. John F. Thompson of Portland, Maine, presided at the meeting, introducing Honorable Percival P. Baxter, Governor of Maine, who made the address of welcome. The other speakers were as follows:

The American College of Surgeons; Scientific Medicine and the Public. Franklin H. Martin, M.D., Director General, American College of Surgeons.

What Scientific Medicine is Doing for the Prospective Mother. John Osborn Polak, M.D., Brooklyn, N. Y., Professor of Obstetrics and Gynecology, Long Island College Hospital.

Experimental Medicine and its Relation to Public Health. Fred Bates Lund, M.D., Surgeon-in-Chief, Boston City Hospital.

The Cancer Problem in New England. Frederick L. Hoffman, M.D., Third Vice President, Prudential Life Insurance Company of America.

Hospital Standardization and the American College of Surgeons. Malcolm T. MacEachern, M.D., C.M., Ottawa, Canada.

Better Hospitals. Rev. C. B. Moulinier, S.J., President, Catholic Hospital Association.

How You Can Aid Your Hospital. Mr. Robert Jolly, Superintendent of the Baptist Hospital, Houston, Texas.

Clinics were continued on the second day, May 16, and the meeting closed with a scientific session presided over by Dr. Edville G. Abbott, chairman of the Maine executive committee. The program for this meeting follows:

Pelvic Inflammation. John Osborn Polak, M.D., Brooklyn, N. Y.

The Relations between the Surgeon and the Medico-Legal Pathologist. George Burgess Magrath, M.D., Boston, Massachusetts.

Certain Problems in Connection with Fractures of Bones. Charles L. Scudder, M.D., Boston, Massachusetts.

MARITIME SECTION

No more enthusiastic meeting of the College has ever been held than that of the Maritime Section, which convened in Halifax on May 19 and 20. Fellows of the College from all the Maritime Provinces were in attendance, together with a number of medical men drawn from Nova Scotia, New Brunswick, and Prince Edward Island.

A carefully prepared program of clinics at the Victoria General Hospital, Camp Hill Hospital, and the Children's Hospital occupied the Fellows on the mornings of May 19 and 20.

A large representation of hospital people at the School for the Blind Friday afternoon made the hospital conference one of unusual interest. The same topics were discussed at Halifax as in Portland, with the exception that Dr. Arthur Morse, professor of obstetrics and gynecology of

the Yale Medical School, spoke on "Hospital Standardization from the Surgeon's Standpoint."

The round table discussion, led by Dr. MacEachern, gave rise to an exceedingly valuable exchange of ideas regarding hospital problems and conditions in Maritime Canada.

The School for the Blind was crowded again at eight o'clock in the evening, when Dr. H. K. MacDonald, chairman of the Nova Scotia Section, introduced His Honor McCallum Grant, Lieutenant-Governor of Nova Scotia, who welcomed the visiting surgeons to the Province.

The program for the meeting follows:

The American College of Surgeons; Scientific Medicine. Franklin H. Martin, M.D., Director General, American College of Surgeons.

What Scientific Medicine is Doing for the Prospective Mother. Arthur Morse, M.D., New Haven, Connecticut.

The Work of the American Society for the Control of Cancer. John Stewart, M.B., C.M., Halifax.

Better Hospitals. Rev. C. B. Moulinier, S.J., President, Catholic Hospital Association.

Hospital Standardization and the American College of Surgeons. Malcolm T. MacEachern, M.D., C.M., Superintendent, Vancouver General Hospital, Vancouver, B. C.

How You Can Aid Your Hospital. Mr. Robert Jolly, Superintendent, Baptist Hospital, Houston, Texas.

At two o'clock Saturday afternoon a scientific session was held at Dalhousie University with the following speakers:

H. K. MacDonald, M.D., Chairman, Presiding.

Infections of the Knee Joint. Ross Miller, M.D., Amherst, N. S.

The Actual Cautery. John Stewart, M.B., C.M., Halifax, N. S.

An elaborate dinner given at the Halifax Club to the Fellows of the College closed the session. An executive committee for the ensuing year was elected for the Maritime Provinces, and, by unanimous vote, St. John was selected as the next meeting place.